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MENINGITIS, SINUS THROMBOSIS AND ABSCESS OF THE BRAIN

MENINGITIS, SINUS THROMBOSIS AND ABSCESS OF THE BRAIN

WITH APPENDICES ON

LUMBAR PUNCTURE AND ITS USES

AND

DISEASES OF THE NASAL ACCESSORY SINUSES

BY

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AUTHOR OF 'TUMOURS OF THE CEREBELLUM'

LONDON

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TO THE MEMORY OF

PROFESSOR SIR THOMAS McCALL ANDERSON,

M.D., F.F.P.S. Glas.,

IN RECOGNITION OF HIS EMINENCE AS A PHYSICIAN AND

CLINICAL TEACHER, AND HIS WELL-MERITED

POSITION OF

THE STUDENTS' FRIEND,

THIS WORK IS DEDICATED BY A FORMER PUPIL

603291

PREFACE

THIS work is written with a desire to place before the reader in a small volume a consideration of diseases which in their earlier stages often exhibit a striking similarity of symptoms, and to differentiate between them as far as may be possible.

The book has exceeded the limit originally marked out for it. For example, the frequent reference to lumbar puncture in the text seemed to lead inevitably to stating how normal cerebro-spinal fluid is constituted, and the changes it may undergo in disease of the central nervous system; the use of lumbar puncture as a diagnostic and therapeutic agent necessitated attention being directed to spinal anæsthesia.

Similarly, diseases of the nasal accessory sinuses were so often mentioned as possible causes of cerebral abscess and sinus thrombosis that symptoms of the causal diseases required some prominence.

The cases in the lists have been for the most part

collected from the pages of current medical literature—chiefly the *Lancet* and *British Medical Journal.*

I desire to express my thanks to my neighbours, Drs. Appleton and Robinson—the former for courteously placing at my disposal an interesting case which occurred at the Sculcoates Union Infirmary, and the latter for supplying me with notes of a case which occurred in his private practice.

JOHN WYLLIE.

262, BEVERLEY ROAD, HULL,
December, 1910.

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MENINGITIS, SINUS THROMBOSIS, AND ABSCESS OF THE BRAIN

INTRODUCTORY NOTES

CAUSAL DISEASES

THE author, having in mind the supremely important place which causal diseases hold in the matter of diagnosis, thinks it will not be out of place to devote the first page of this work to reminding the reader that when symptoms suggestive of endocranial disease arise during or shortly after another ailment, the form of the primal disease points with considerable certainty to the nature of the endocranial disease present, although by doing so he may be charged later on with reiteration.

If the patient be an adult, and suffers from old tuberculous disease of the lungs, bowels, bones, joints, or glands, or, if a child, he suffers from caseation of the peribronchial, mesenteric, or cervical glands, and the two former in particular, tubercular meningitis is the variety of brain trouble most likely to be present; and non-tuberculous diseases of the lungs—such as bronchiectasis, putrid bronchitis, empyema, or badly resolving pneumonia, and malignant endocarditis—are of equal importance as indicators of brain abscess.

Otorrhœa, recent or chronic, and suppuration in the accessory nasal cavities, may mean brain abscess, sinus thrombosis, or suppurative meningitis, or any two or all of them in association. Scarlet fever and measles (when there is no aural discharge), pneumonia (during the height of the disease or when convalescence is proceeding), rheumatic fever, influenza, and smallpox, are for the most part productive of suppurative meningitis; but it may be abscess in case of pneumonia, influenza, and smallpox. A recent confinement (mostly when there has been obvious septicæmia, and also when the woman has seemed to do well), the anæmia or chlorosis of young girls, cachexia and great weakness, erysipelas, a long-continued diarrhœa in infants, and boils or pustules on the face, all point in the greatest number of instances to thrombosis of the cavernous and longitudinal sinuses. For example, supposing one had a case (not otitic) exhibiting symptoms chiefly meningeal, with slow pulse and several degrees of fever, but there were superadded motor aphasia and right-sided hemiplegia or paresis, the question would arise, Is the case meningeal, suppurative or tuberculous, with a thick exudate over the left Broca's convolution, or a cerebral abscess at the same site, plus meningitis? The nature of the causal disease, if any, would be of inestimable value in enabling us to arrive at a correct conclusion, particularly if an examination of the cerebro-spinal fluid had afforded no assistance. The fact that the patient had not suffered recently from an acute febrile disease would, to a great extent, rule out suppurative meningitis, while tuberculous disease would indicate

tubercular meningitis, and any pulmonary disease not tuberculous would just as forcibly point to cerebral abscess, with probably an associated meningitis.

Kernig's Sign.

In brief, Kernig's sign consists in a difficulty, when sitting up, to keep the legs and thighs in a straight line. It may be elicited by placing the patient squarely on his back, with his legs relaxed and fully extended at the knees; then, the leg being bent so as to bring it to a right angle with the trunk, flexion also takes place at the knee; or, on bringing him to a sitting posture, the thighs become flexed on the abdomen and the knees are bent; the legs cannot in either case be extended without using violence, on account of the contracture of the posterior muscles of the thigh. If the patient sits upon the edge of the bed, and an attempt is made to bend the leg upon the thigh, there is a similar condition of contracture.

Kernig first described this sign in 1884, and he suggested that it was only present in affections of the pia mater, and always in inflammation of that membrane.

Its diagnostic value as a symptom of cerebral meningitis is great, but it is by no means an unequivocal sign. It is certainly present in the great majority of cases of tubercular meningitis—*i.e.*, in from 85 to 90 per cent.; it has also been found in lateral sinus thrombosis when there was no meningitis, and also in hæmorrhage into the subarachnoid space, the symptom becoming possible by the thrombus acting as an irritant of the

meninges; and in cerebral abscess, when the latter is near the surface, it acts similarly to the clot.

Moreover, Kernig's sign has been found in pneumonia, and Netter, out of 314 cases of typhoid fever, found it present in 44 of them, some of which exhibited other symptoms of meningitis, while others did not. He found that its presence in typhoid fever betokened a serious case, the relapses and mortality being three times greater than in those in which it was absent.

It has been found absent in proved cases of meningitis, and present in diseases other than those already named, such as pleurisy with effusion, vertebral rheumatism, hysteria, and sciatica, in which no nervous disease was demonstrated at the autopsy; it has also been found in many chronic nervous diseases in which the cerebral meninges were not diseased.

It may pass away as the disease advances. Such passing away is said to be an attribute peculiar to epidemic cerebro-spinal meningitis.

The sign may occur in the upper extremities, as well as in the legs, as a very early symptom of cerebral meningitis.

The following signs have been found present in cerebro-spinal meningitis:[1]

The Sign de la Nuque.—To obtain this reflex the neck is bent forward, and it is found that the lower limbs at the same time become flexed at the knee and on the abdomen; present in 97 per cent.

Babinski's Sign.—Passive flexion or extension of one lower limb provokes a similar movement on the opposite side in about 60 per cent. of cases.

[1] *British Medical Journal*, Epitome No. 1, July 2, 1910.

CHAPTER I

MENINGITIS

THE arachnoid mater and pia mater, the leptomeninges (λεπτός, thin; μήνιγξ, a membrane), form together what is known as the pia-arachnoid membrane, because any disease which affects the one involves the other, and inflammation of this double membrane we speak of as meningitis or leptomeningitis. The arachnoid is a non-vascular membrane of fibrous tissue of extreme thinness which invests the brain, passing from one convolution to another, bridging over the sulci and fissures other than the longitudinal. The pia mater is a thin vascular, fibrous tissue membrane, consisting of a network of bloodvessels, held together by fine areolar tissue. It closely covers the outer surface of the whole central nervous system, dips into the sulci and fissures, and, as the **velum interpositum**, projects into the ventricles; and it gives off from its under surface fine vessels, which penetrate into the substance of the brain. From the fact of the pia mater dipping into the sulci and sending vessels into the brain, we find the explanation of the constant association of meningitis and encephalitis, and in the free nervous supply of the investing membrane from the sympathetic and several cranial nerves we recognize one way in

which pain may be caused, either by inflammation from within or from pressure outside the skull.

Meningitis is popularly known as inflammation of the brain, and while, strictly speaking, this is somewhat of a misnomer, it is not, for the reasons already adduced, altogether incorrect; in the double word 'meningo-encephalitis' we should have a happier description, but a more unwieldy title.

The space between the dura mater and the arachnoid is known as the subdural, and that between the arachnoid and pia mater as the subarachnoid, space.

Many, perhaps the great majority, of the symptoms of meningitis are common to all the varieties of it, through being caused by increased pressure within the skull, and, apart from the information gained by lumbar puncture, the doctor has his first and greatest assistance in distinguishing between them in the knowledge of their probable causal diseases. For the same reason many of the symptoms to be brought under notice in intracranial abscess and cerebral sinus thrombosis are also the symptoms commonly observed in meningitis, and they may also be seen in the early stages of pneumonia and typhoid fever when no intracranial disease exists.

Writers have divided cerebral meningitis into many different kinds, one differentiating as many as twenty-five—a finesse in diagnosis that is not practical, not necessary, and will certainly not be attempted in these pages.

From its site, meningitis has the following descriptions: When occurring chiefly at the base, 'basic'; at

the vertex, 'vertical'; when only in connection with the hind-brain, 'posterior basic'; in the ventricles, 'ventricular' or 'occlusive'; 'cerebro-spinal'; and 'general.'

If we classify it according to the nature of the causal micro-organism, we have:

Tubercular meningitis, when it arises from Koch's bacillus.

Posterior basic meningitis and epidemic cerebro-spinal meningitis (which differ slightly in their clinical manifestations), when it arises from *Diplococcus intracellularis.*

Acute suppurative meningitis, when it arises from staphylococci, streptococci, etc.

Syphilitic meningitis, when it arises from *Spirochæta pallida.*

Tubercular Meningitis.

As **tubercular meningitis** is the intracranial disease which the doctor in general practice sees more frequently than any other, it will be considered in this work first, and to some extent be taken as a type, and other meningeal diseases will be studied as to the manner or degree in which they differ from it. It is also termed **granular** meningitis, on account of the 'grey granulations' which are found on the cerebral pia mater and elsewhere, and is frequently called 'basal meningitis,' because tubercle in very many cases is only observed on the meninges and fissures at the base of the brain; but it is wrongly so called, as tubercle has been seen often enough—by the writer on one occasion—on the meninges of the vertex. To a past generation it was known as **acute hydrocephalus,** because of the

considerable dropsy of the lateral ventricles, which seemed to the medical mind of the time to exceed the tuberculous deposits in importance; but such a condition is common also to non-tuberculous forms of meningitis.

Age.—This disease is said to occur at any age from six months to sixty years or more, but most cases are seen between the ages of one and five years, or at any rate in early childhood. The last six cases under the author's care were aged as follows: Female, one year eight months; male, three and a half years; female, five years; male, six years; male, twelve years; male, twenty-four years.

Ætiology.—It is the established opinion that tubercular meningitis is not an independent affection, but only part of a general acute tuberculosis, or is an auto-infection from tubercle in the white substance of the brain, or a direct extension from a tuberculous deposit in its cortex; and the pia-arachnoid, being the most delicate structure affected, and the most seriously so, often develops symptoms earlier than the other organs or tissues of the body which have a similar liability to tuberculosis, and death takes place before any other part produces symptoms of disease. A few writers dissent, and hold that the meningeal disease may be primary. The sequence of events is that there is tuberculosis, in one or more distant organs, of older standing than, and causative of, the meningeal disease, in that it yields a focus which is carried to the vessels at the base of the brain, and which may or may not have been observable before the oncoming of meningeal

symptoms. In adults it is old tubercular disease of the lungs, bowels, bones, and joints; in children it is very frequently the spleen or a caseous peribronchial gland, less commonly a mesenteric, and only rarely a cervical gland, the lungs and bowels, etc., probably being healthy. Two or three members of the same family may suffer simultaneously, or with intervals of months or years between. The writer has witnessed three children of the same parents suffer as they approached the age of three years.

Tubercular meningitis is very likely to occur in those who suffer from any of the above diseases and are at the time ill-nourished, live in insanitary homes, or whose general tone has been affected by much brain-work or a lowering disease like measles or whooping-cough.

It is beyond question that the deposition of tubercle upon the cerebral meninges is accelerated or developed through violence, the patient being probably already tuberculous. Most medical men can point to cases which, within their personal knowledge, have developed brain symptoms so quickly, probably within a day or two, after a fall, blow, or simple box on the ears at school, as to give rise to the opinion that the two were in relation of cause and effect, and the tuberculous nature of the disease has been in such cases often verified post mortem by eyesight evidence and bacteriological examination. A case occurred in which all the parties were well known to me, but I was not in medical charge. Neither parent had been too gentle in the treatment of a two-year-old little girl, and on her sickening and dying one charged the other with having

caused her death. At the necropsy tubercle was found on the basal meninges, and death from natural causes was the conclusion arrived at. The medico-legal aspect of the case was thus settled, but how far possible traumatism was concerned in causing the disease to develop remained doubtful.

Anatomical Changes and Pathology.—Tubercular meningitis is characterized by the presence of small tubercles, seldom larger than a millet-seed, and therefore called 'miliary,' which are gelatinous in consistence and grey in colour, and on the latter account are called 'grey granulations.' They are situated upon the surface of the pia mater, in the course of and surrounding and infiltrating the **tunica adventitia** and the entire walls of its smaller vessels, and within their perivascular sheaths; they occur chiefly at the base of the brain, and are there more numerous in the subarachnoid space, where the large lymph sacs are located in the circle of Willis, in the Sylvian fossæ and fissures, on the surface of the pons Varolii, and the under surface and sides of the cerebellum. They may also be seen, but much less often, upon the sides of the cerebral hemispheres, especially at the bases of the ascending frontal and parietal convolutions; upon that part of the under surface of the brain which rests upon the orbital plate of the frontal bone; in the sulci of the vertex; on the under surface of the dura mater; on the walls of the ventricles; and sometimes on the spinal meninges, may be as far as, and including, the cauda equina, chiefly over the inner surface of the dura, and in the pia over the lumbar enlargement.

Inconsistent though it may be with the name of the disease, there may be no tubercles present—that is, observable by the naked eye—either upon the meninges or in or upon the brain, although the exudate at the base may have the orthodox appearance, and the tubercle bacillus be detected in it and lymphocytes in the cerebro-spinal fluid, and diffuse miliary tuberculosis be found in the lungs or other organs.[1] Probably the microscope in such cases would reveal the commencement of the process of tubercle deposition. The tubercles consist of mononuclear leucocytes, otherwise lymphocytes, and polymorphonuclear cells, the former being in great excess in most cases while the tubercles are still young, but as they get older the lymphocytes decrease in numbers and the polymorphs increase. This cellular infiltration of small vessels may exist without inflammation, but generally quickly develops it, and, as a consequence, the arachnoid membrane loses its glistening appearance and becomes sticky to the touch; the pia mater becomes glued to the surface of the brain, and cannot be stripped from it without taking with it portions of brain substance; the large lymph sacs become distended with a fibrino-serous effusion, in which are imbedded the second, third, and fourth cranial nerves, to which the important clinical symptoms of optic neuritis, ptosis, pupillary abnormalities, and squint, so often presented to us in tubercular meningitis, owe their origin. A further result of inflammation is the deposition over the greater part of the base of the

[1] *Vide* case recorded by Dr. Bigge in *British Medical Journal*, May 15, 1909.

brain of an exudate or tough layer of opaque, white, or yellowish-green lymph; it is rarely seen on the convolutions at the vertex, which become dry and flattened by pressure, and there may be a little on the cervical portion of the spinal meninges. If the exudate is thick over the motor area of the brain, it may cause hemiplegia, monoplegia, twitchings of the face, etc.; and if copious over Broca's convolution in the left hemisphere, aphasia may result. The exudate consists of young tubercles, which develop out of the cellular overgrowth. The free portion is coagulable, and in the more recent cases is chiefly fibrinous. In it may be present Koch's tubercle bacillus, with or without cells; but cells, chiefly lymphocytes, may be found without the bacillus. All meningeal exudates appear to the naked eye very much alike, and may be produced by any of the infective micro-organisms.

Along with these changes there may be moderate effusion into the ventricles of a fluid which is often clear, occasionally turbid, and very infrequently purulent, and which may be looked upon as the probable cause of the headache so often present; thrombosis of the veins of Galen, and occasionally of one or more cerebral veins, or of the superior longitudinal sinus, and isolated patches of encephalitis. The brain substance is highly vascular from the hyperæmic condition of the vessels near the tubercles.

When the tuberculous deposits are in the convolutions or other parts of the brain, the corpus striatum is an occasional site; their breaking down causes cerebral abscess, but the special symptoms of abscess in any

particular site or of the thromboses are lost in those of the meningitis.

As regards the corpuscular elements of the blood, the count of red cells is rather high. In 60 per cent. of cases there is no increase of white cells, but there may be increase of the lymphocytes in favourable cases, and the polymorphonuclears may also at times be increased.

The cerebro-spinal fluid is clear, but may become milky on agitation, or it is turbid, and may or may not contain Koch's bacillus, but contains albumin, and is therefore coagulable, fibrin, and abundance of lymphocytes, and some polynuclear leucocytes: occasionally the latter are in excess of the former. The fluid is under considerable pressure, almost twice as much as is the case in health, 120 to 180 millimetres being common.

Far from exhibiting the sequence of symptoms about to be described, these present may deviate so far from those usually noticed in tubercular meningitis as to cause us to form an opinion widely divergent from the correct one. The case, if in a child, may be very acute, running rapidly from headache, vomiting, and restlessness, to convulsions, coma, and death; if in an adult, after a short period of vague discomfort, violent headache and noisy delirium suddenly develop, and continue until the patient dies from exhaustion. Such a sequence of matters occurred in a young single woman in Hull as these pages were being prepared for the press, and on post-mortem examination the disease was found to have been tubercular meningitis. Symptoms of tubercle on the brain or its meninges may be modified

or overshadowed by those of some associated disease, which seems to explain the symptoms.

In babies and those who have long suffered from phthisis sleepiness may only be noticed. In adults there may be only loss of appetite, a general out-of-sorts condition, or mental dulness, with possibly some abnormality of the knee-jerks; or symptoms which refer one to a definite area of the brain may only be present. The disease may run its course in a few days or last through months, but the usual duration is about three weeks; the patient may die well within a week if he is suffering from obvious tuberculosis elsewhere.

Tubercular meningitis is considered to consist of three stages, the transition from one to another being gradual and giving no special sign.

1. **The Stage of Invasion or Irritation.**—This stage may begin by a general feeling of **malaise**, or severe frontal headache, which in the case of young children finds forcible expression in the frequency with which the patient applies its hand to its head, frequent and apparently causeless vomiting and constipation, and there may be convulsions in children and delirium in older people. The pulse is quick, and there may be two or three degrees of fever; the pupils are contracted, and it is noticed that the skin is unduly sensitive to the touch, the hearing is less acute, and that the patient avoids the light, turning to the wall, becomes drowsy, is restless and fretful, and irritable when disturbed; and there may be noticed some rigidity or stiffness of the nuchal muscles.

2. **The Stage of Pressure.**—The drowsiness in-

creases, and the child may seem unconscious; the vomiting becomes less or ceases entirely; the constipation continues; and the abdomen is retracted, its appearance being so characteristic that the special names of boot-shaped, navicular, carinated, and scaphoid, have been used to describe it. The temperature may fall and the pulse drop below normal in the number of its beats, and very likely be irregular; the vaso-motor condition of the skin is raised, and we recognize this fact by the **tâche cérébrale**, otherwise **tâche méningique**, or red linear mark, produced by drawing the finger-nail sharply across the face, limbs, or abdomen, being easily obtained and persisting, and herpes and erythema may also be seen. Kernig's sign is almost always present, and there may be paralysis of an arm or leg, or of both arm and leg, and there are very likely to be drooping of one or both upper eyelids, convergent strabismus, and less often facial paralysis. The head becomes a little more retracted, or the patient rolls it uneasily from side to side, and the muscles of the neck are more rigid; and if the patient is a child he screams out suddenly with what is known as the 'hydrocephalic cry.' The breathing is irregular and sighing; optic neuritis is observed in the distended and tortuous state of the retinal veins and contracted arteries, small hæmorrhages in the retina, and afterwards swelling of the discs, with more or less blurring of their edges, choked discs being less commonly noticed than optic neuritis. The pupils are now dilated, and probably unequal and insensitive to light, or they alternately contract and dilate—the **instabilité pupilaire** of the

French writers—or they exhibit the 'paradox reflex,' contracting in the shade and dilating on exposure to light.

3. **The Stage of Coma and Paralysis.**—A typhoid state now supervenes, with dry, glazed tongue; diarrhœa takes the place of constipation; the patient loses power over his sphincters, and passes urine and fæces involuntarily; and if paralysis has not yet occurred, it appears. The pulse and temperature again rise, the latter reaching as high as 105° F. or falling to subnormal, possibly to 94° or 95° F.; the drowsiness has passed into stupor and that into coma, and the patient cannot be roused; the optic neuritis has become intense, and he may be blind, and therefore no longer avoids the light, but keeps his eyes wide open and staring, and with the ophthalmoscope tubercles may, but not always, be seen on the choroid coat of the eye. There may be nystagmus; convulsions are frequent; the pupils are widely dilated, and no longer respond to variations of light; and the fontanelles are raised and pulsate. Cheyne-Stokes respiration is the rule, and the temperature may rise before death to 106° or 107° F.

In any stage of the disease the temperature may rise as high as in any other form of cerebral meningitis.

The opinion that inequality of the pupils is always present in tubercular meningitis is altogether incorrect and misleading, the opinion probably being arrived at through the frequency of inequality. Dilatation of the pupils is due in these cases to intracranial pressure; but if there is more of such pressure on one side than the

other, on account of a greater deposition of tubercle and exudate, inequality results, but it is a matter of accident.

The following case will be seen to embody many of the above conditions:

F. M., aged three and a half years, was brought to me on the evening of April 3, 1908, and his mother said that he had been out of sorts a few days, and she thought he had a sore throat, which surmise, on examination of the throat, proved to be incorrect.

On April 6 I saw the child at his own home, and was informed that he had cried out suddenly during the night as if he had been frightened, that he often raised his hands to his head and put his fingers into his ears, and that since I saw him three days before he had vomited frequently, had had no action of the bowels, and had complained that his head hurt him. I noticed that his hearing was not very acute, and that if left to himself he dozed. His temperature was 100° F., and pulse 80 beats per minute and regular; his breath was offensive, and tongue slightly coated. The tâche cérébrale was easily elicited, and the abdomen slightly retracted, but there were no spots. The family history was distinctly tuberculous: a baby brother had been born with what was probably tabes mesenterica—that is, he had a hard abdomen, and lumps in it which suppurated—and from which he died within a month of his birth; one sister and two maternal aunts of the mother had died from consumption, and one of her brothers had long suffered from enlarged cervical glands and abscesses.

April 7: Pulse 80; temperature 99·2° F.; abdomen more distinctly retracted, and decidedly boat-shaped. Knee-jerks were absent; there was no ankle-clonus, and Kernig's sign was well marked; the breathing was shallow, and the pupils were dilated and slightly unequal, but reacted sluggishly to light.

April 9: Pulse 120; temperature 102° F.; respirations were thirty-six per minute, and of the Cheyne-Stokes type, consisting of three or four deep and strong respirations, followed by four or five weak and feeble ones. Up to this date he had avoided the light, and when facing it his eyes were closed; now he could lie facing it with his eyes wide open and fixed; the pupils were dilated, unequal, and irresponsive.

April 10: Pulse 120; temperature 102° F.; respirations as described above; had four or five convulsions during the night, and the drowsiness had passed into stupor. Was very restless during ophthalmoscopic examination, so that the state of the discs could not be ascertained; but it was noticed that the retinal veins were much distended, and that there were no retinal hæmorrhages. There was also now right-sided hemiplegia, which continued to the end.

April 11: The pulse, temperature, and respirations were the same as on the two previous days. There had been several fits during the night; the face was flushed; there was no retraction of the head at this time or at any time during the illness. The patient had refused food and medicine for two days, and could not be roused, and was apparently deeply comatose.

April 12: Pulse 140; temperature 100° F.; Cheyne-Stokes respirations; frequent convulsions during the

night; still comatose, and died early next morning in a fit.

Diagnosis.—Of all the symptoms that may be presented by a person suffering from tubercular meningitis, there are none peculiar to that disease only, certainly none of the subjective and few of the objective ones, the chief of the latter being the presence of Koch's tubercle bacillus in the cerebro-spinal fluid, and tubercles on the choroid or vascular tissue of the eyeball; but both may be absent in the particular case one is doubtful about. Several of the symptoms mentioned above are highly suggestive of tubercular meningitis, but most of them are also to be seen in other forms of meningitis, and many in diseases of a totally different kind. It is their association that enables one to refer them to tubercular meningitis, and especially is this so when the patient belongs to a tuberculous family, or is himself tuberculous.

The feelings of malaise and headache are premonitory to many ailments, but the avoidance of light is as a guide-post that fortunately stands in our way, and we cannot attach too great an importance to it as suggesting meningeal inflammation. A Hull physician, now deceased, in consultation on a little boy who for three days had had headache, persistent vomiting, and a very rapid pulse, but who at the moment was looking towards the window, said to the author: "If your patient can steadily face the light in the first days of his illness, no matter how much headache there may be or have been, no matter how high or low the temperature may be, or how quick the pulse, it is very certain that you are not dealing with a case of cerebral

meningitis;" and he was right in that particular case, as it afterwards came to light that the child had had a surfeit of chocolate, which had been supplied by unwise friends before he started to travel alone from London to Hull. Such a conclusion, however, advances us only one step along the road: if the patient cannot, when he is suffering from headache, etc., tolerate the light, the intolerance is only suggestive of meningeal irritation, not to that of tubercular meningitis in particular.

Hyperæsthesia of the skin is said to be more distinctly suggestive of tubercular than any other form of meningitis, but it is frequently absent. The tâche cérébrale is still less determinative, for we observe it in all those states in which the vaso-motor condition of the skin is active, and it is easily obtainable in typhoid fever, and often in cerebral abscess. Optic neuritis is common to all acute intracranial diseases, as well as those which run a subacute or chronic course, as do tumours, and it has been noticed in typhoid fever, anæmia, and I believe in hysteria. The state of the pupils may guide us to the stage of the disease, but neither the pupils, ptosis, strabismus, or any other form of paralysis, labels the case as tubercular meningitis. Tubercles in the choroid are definite as a symptom of tubercular meningitis **only** when other meningeal symptoms are also present, and very frequently the choroid is free from them.

Retraction of the head and neck and rigidity of the nuchal muscles are even less reliable than any of the foregoing symptoms; they may be seen in some cases of acute suppurative meningitis, most cases of cerebellar bscesses and tumours, and they are still more likely

to be seen, and in a much more severe degree, in epidemic cerebro-spinal and post-basic meningitis, and in pachymeningitis of the cerebellar fossa. The boat-shaped abdomen is common to other forms of cerebral meningitis, and in any case it is not well marked until the disease is advanced, and the sighing respiration is only suggestive of intracranial pressure upon bulbar centres.

Skeer's sign of a circlet of tubercles on the irides near their free margins is said to be pathognomonic, but it is seldom to be seen, and disappears again in a few days. The hydrocephalic cry, if it did not occur with other cerebral symptoms, might easily be looked upon as the night terrors exhibited by children suffering from worms or disordered digestion.

However definitely Kernig's sign may suggest meningeal trouble, it is certainly not peculiar to tubercular meningitis, and in a small majority of such cases (some writers say half of them) it does not occur at all; besides, it occurs in other forms of meningeal inflammation, in sinus thrombosis and meningeal hæmorrhage, and is an outcome of irritation of the meninges rather than of their inflammation. Babinski's extensor reflex of the great toe is said to be more common than Kernig's sign. Urticarial weals have been seen on the extensor surfaces of the arms.

If the pathological condition be localized and the exudate be thick over certain areas, localizing symptoms may obsess one's mind so strongly that a tentative diagnosis only is possible.

Prognosis. — Tubercular meningitis is almost uniformly fatal, but alleged recoveries are not infrequent, as in cases where persons have exhibited meningeal

symptoms and recovered, and after a time have died from accident or other diseases, and on post-mortem examination tubercle has been found on the basal meninges and in the Sylvian fissure, and very likely also caseous peribronchial glands and miliary tubercles in the lungs and other organs. In other cases two children of the same family have been ill at the same time with identical cerebral symptoms; one has recovered and the other died, and in the latter case tubercle has been found on the pial vessels.

Symptoms may totally disappear, and the child for weeks, months, or more than a year, may seem to be in his ordinary health, taking his part in play, or attending school; yet the disease has only intermitted, and there may be more than one intermission before the last and fatal attack.

The lookout is wellnigh hopeless, but with recoveries estimated at 2 per cent., and still more frequent long intermissions, we are justified in persevering with treatment—at any rate, in the early part of the disease. But the author feels bound to say that out of the many cases that have come under his notice during more than a quarter of a century of practice, he has seen neither any case of recovery nor intermission; slight improvements during the illness—sufficient, indeed, to raise hopes—there have been, but they always ended in disappointment.

Treatment.—Treatment is prophylactic, in so far as it applies to keeping your weakly patients in good sanitary surroundings, and the removal of such possible foci of infection as tuberculous glands. When the disease is once established, quietude is above all else

essential, promoting the patient's comfort, if not assisting towards his recovery; he should therefore be kept in a darkened room, and visitors be excluded. The ice-cap to the head may relieve the headache, and for the same purpose analgesics may also be given. Milk, with soda-water, or predigested with peptonized powders, may be the only food the stomach will retain, and if the vomiting persists, rectal feeding only should be continued. Koch's tuberculin should certainly have a trial, administered according to von Pirquet's method by rubbing it into scratches in the skin. Lumbar puncture may be performed and repeated at intervals if found to give relief. Medicinally, mercury is the one drug to which the most eminent medical authorities ascribe any power, and it may be given both by inunction and mixture. Of the latter a baby one year old may take 2 minims of the liq. hydrargyri perchloridi, with 1 grain of the iodide of potassium and 2 grains of the bromide of potassium if there is much restlessness, every four hours. Syrup of phosphorus has been highly extolled, in the strength of $\frac{1}{6}$ grain to 1 ounce; 2 minims for every year of age should be given every four hours. Iodoform ointment, 15 grains to 1 ounce of lanoline, rubbed into the scalp and back of neck every eight hours, is favourably spoken of by some who have had the good fortune to see recoveries.

Acute Meningitis.

Acute meningitis (idiopathic simple meningitis, microbial meningitis, suppurative cerebro-spinal lepto-meningitis, non-tubercular meningitis) may be purulent from the first, begin as serous and become purulent,

be localized over a small area of the brain or be diffused over the greater part of its surface.

Definition.—Acute meningitis is a simple non-tubercular inflammation of the pia mater of—(*a*) the convexity of the brain, the site in the exanthemata and other acute febrile diseases; (*b*) the sides and base; (*c*) the base only, which is the form met with in otitis media. It is, however, a disease for the most part of the convexity, as tubercular meningitis is a disease of the base, of the brain. In addition to the cerebral pia mater, the ventricular cavities and the whole length of the spinal meninges may be affected, as well as a small extent of the cranial and spinal nerves.

Ætiology.—Acute meningitis is a disease seen more frequently in males than females; and while it may arise spontaneously in cachectic or broken-down persons, or those who have been subjected to long exposure to the sun (insolation), its chief causes are suppuration in the middle ear, the path of infection being directly through bone, or by the sheaths of the facial and auditory nerves, suppurative phlebitis, suppuration in the accessory nasal cavities and the orbit, erysipelas of the head and face, anthrax, from the rupture of a cerebellar or cerebral abscess into the subarachnoid space, as took place in three cases recorded under these headings; or a brain abscess may cause meningitis without rupture, as was the result in one case. It may occur during the progress of, or soon after, pneumonia, puerperal septicæmia, scarlet fever, influenza, typhoid fever, smallpox, measles, ulcerative endocarditis, acute rheumatism, and cases have been recorded as caused by gonorrhœa. It may follow severe mental shocks, mental

exhaustion from overstudy, as suppurative meningitis from the beginning in case of pyæmia, and has been known to follow the excision of a suppurating eyeball, the removal of an aural polypus, a suppurating vermiform appendix; or there may be two probable causal diseases, as in the case of pneumonia and septic endocarditis, creating a difficulty as to which the meningitis is to be ascribed.

Anatomical Changes and Pathology.—The first change from the normal is a hyperæmia of the pia-arachnoid. The small vessels, particularly the veins, of the pia mater of the convexity are loaded with blood, and, partly from this cause and partly from an exudation of leucocytes into their perivascular sheaths, become tortuous, swollen, and of a deep red colour. A little later the arachnoid becomes opaque, and beneath it is formed a sero-fibrinous fluid, pus, or lymph-like matter, liable to cohere and form layers. Fluid may be increased in quantity in the subdural or subarachnoid space (external hydrocephalus), and there it may be serous, sanguinolent, or purulent, and the lining membranes of the ventricles, which may be greatly distended (internal hydrocephalus) with a similar fluid, may be rough and congested. When pus is effused at the base, it is not as a rule diffuse, but contained in the large lymph sacs in the subarachnoid space, mentioned in tubercular meningitis. The largest of these sacs is upon the under surface of the cerebellum, and is wanting in the trabeculæ common to the smaller ones; there is intercommunication between each, and from them to the spinal cord. There may be minute hæmorrhages into the substance of the nerves which are paralyzed, the

hæmorrhages perhaps being caused by the infective micro-organisms which travel along their perineural sheaths, and the nerve sheath or trunk may be inflamed, or the latter undergo degenerative changes. The inner surface of the dura mater and the cerebral cortex are also reddened, and the latter may be soft, but the pia mater strips off it easily. If there is great effusion into the ventricles, the convolutions of the brain are dry, perhaps flattened, and the sulci shallow. When the inflammation is very diffuse, the pia mater of the upper part of the cord is also reddened, and when purulent the condition may extend the whole length of the cord; and it is very likely to do so in the acute meningitis following otitis media, suppuration in the accessory nasal cavities, and influenza. There is also a tendency to thrombosis of the veins emptying into the superior longitudinal sinus, or the suppuration may be secondary to sinus thrombosis when the latter is due to carious or necrosed bone. In all forms of acute suppurative meningitis the exudate is characterized by much fibrin, and in the early stages of the disease it is chiefly fibrinous. The cells entangled in the fibrin in pneumococcal meningitis are neutrophile leucocytes and mononuclears, but the polymorphs may be in excess, as they also are in streptococcal meningitis. According to the nature of the causal disease, the micro-organism may be the pneumococcus, pneumo-bacillus (Friedlander), streptococcus, staphylococcus, and the *Pyogenes coli communis* has been found. If the patient is much better after the withdrawal of cerebro-spinal fluid, we may conclude that the serous stage has not been passed, or he may die while the effusion is still serous on account

of the toxins being highly virulent. In those cases of acute meningitis which are caused by suppuration in the accessory nasal cavities the orbital plate of the frontal bone is much roughened, and the dura mater of the base is normal, except that portion of it which rests upon the orbital plate, which is thickened.

Symptoms.—The patient—again for the most part a child or young person—if not already suffering from a febrile disease, is suddenly taken ill with intense headache, soon to be followed by a convulsion or rigor, and vomiting, which may be more or less persistent throughout the illness. The temperature is usually 103° F. or 104° F., falling and again rising at frequent intervals, and is likely to reach a high point, 106° to 107° F., shortly before death. The pulse is at first quick, and may remain so, or may fall to 60 or even 50 beats per minute, but again rises before death to 140, 150, or 160 beats.

The breathing is rapid, except when the disease chiefly affects the cerebellar fossa, when it is slow, and towards the end takes on a Cheyne-Stokes character. There is great intolerance of light, and, if the patient lingers, optic neuritis is developed; but the disease is often too quickly fatal for more than some vascular engorgement of the retinal vessels to be recognized. The pupils are contracted in the early stage, but soon become dilated, and soon become unequal and insensitive to light; there is hyperæsthesia of the skin; the head becomes retracted, but not so much so as in post-basic cases. Fits, either unilateral or general, are frequent; the abdomen is retracted; the tâche cérébrale may be easily obtained; the breath is offensive, the tongue coated, and there is constipation until the close

of the illness, when there may be involuntary evacuations. Paralyses in various forms may present themselves as the disease advances. Paralysis of an arm or leg, or both—bespeaking pressure of effused material upon the cerebral motor area—is the type usually seen in meningitis of acute febrile disease, while ptosis and convergent strabismus, pointing to basal pressure upon the third and sixth cranial nerves, are most frequent in the suppurative meningitis of otitis media. The head is always very hot, and if the anterior fontanelle is not yet closed, there is bulging and pulsation. Delirium is frequent until the patient becomes comatose; and one of the great characteristics of the disease is the rapid supervention of coma upon the irritative stage; and if the coma disappears for a short time, delirium is again noticed. Kernig's sign is usually present early. Eight or ten days may be the duration of the illness, but it is often much shorter. Cases occur, especially in children and weakly persons, that seem to want everything that we rely upon as suggesting cerebral meningitis. Kernig's sign, the tâche cérébrale, and retraction of the abdomen, may all be absent; and one only notices that the child is fretful and often crying, showing a tendency to retraction of the head, with pulsation in the anterior fontanelle, and possibly presenting a slight papillitis if the discs are examined.

We may not easily be able to say whether a patient is suffering from tubercular meningitis or acute idiopathic meningitis; we receive only little assistance from the presence or absence of optic neuritis, none from the state of the pupils or pulse, retracted abdomen, tâche cérébrale, photophobia, retraction of the head, fretful-

ness and restlessness, ocular or other paralysis, or Kernig's sign. Distinguished writers say that in many cases it is a matter of the extremest difficulty, and perhaps, indeed, impossible, to distinguish between the two; even lumbar puncture may be sterile in result, and with such expression of opinion the writer must concur.

Purulent cerebro-spinal meningitis, arising from otitis media or suppuration in the nasal cavities—the route of infection in the latter case being *per* the sphenoidal sinus, and through the small bloodvessels in it to the cavernous sinus — may simulate the epidemic form in all particulars, including extreme retraction of the head and opisthotonos, except joint pains and rashes.

Children in whom acute meningitis develops independently of any acute disease or any known cause are said to live longer and have a slightly more hopeful outlook than when the meningitis occurs during the course of or after another disease; but this rule does not seem to hold good in the case of adults, as will be seen from the following case: A man of good family and personal history, thirty-five years old, was taken ill with severe headache and vomiting on June 20, 1883. When seen by me on the following day, he was, to use his own words, 'almost mad with pain in his head'; his pulse was 140, and temperature 104° F. The pupils were dilated, but sensitive, and he could not tolerate light or sound. On June 21 the intensity of headache still continued; pulse 160, and temperature the same as the day before; he was irritable, resented interference, and was at times delirious. On June 30 he was comatose, and died in the evening. There was no paralysis or head retraction.

Otitis Media.—That purulent leptomeningitis and brain abscess are frequently consequent to purulent middle-ear disease we all pretty well know; but it is not equally well known that serous meningitis, the infective process not having perforated the dura, is often due to the same cause. Drs. Barr and Rowan, of Glasgow, who have exhaustively studied the question, say: 'Serous meningitis is a not uncommon complication of purulent middle-ear disease, and may simulate, as regards symptoms, either purulent meningitis or brain abscess. We think that milder forms of serous meningitis occur much more frequently during the course of a chronic middle-ear suppuration than is supposed, and . . . in a patient suffering from purulent ear disease frequent bouts of headache, accompanied by nausea, vomiting, giddiness, or elevated temperature, coming on periodically, are highly suggestive of meningeal hæmorrhage.' They further say that optic neuritis may be present in purulent middle-ear disease when there are no obvious meningeal symptoms; and frequently, when the vascular changes in the fundus do not amount to optic neuritis, there may very often be an engorgement and tortuosity of the veins, with, in other cases, a somewhat pinkish disc.

Pneumonia.—Meningitis is not a frequent accompaniment of pneumonia. Dr. Aufrecht, writing in Nothnagel's 'Encyclopædia,' found only seven cases of meningitis in 253 cases of fatal pneumonia; but, still, it occurs more frequently during pneumonia, or, rather, after the crisis has passed, and the patient seems to be doing well, than in connection with any other acute febrile disease, except, perhaps, scarlet fever. There is

some diversity of opinion as to how the infective micro-organism reaches the brain. Huguenin, of Munich, favours the opinion that purulent foci from the pneumonic exudate are carried by the blood-stream to the vessels of the brain. This may be, and probably is, the case in the developed disease and during the process of resolution; but when resolution is to all appearance complete, other explanations become necessary. Weichselbaum says the pneumococci reach the meninges by the cellular tissue of the neck. The first notice that some complication has arisen is that the patient complains of headache when there was none previously, or, if there was, it is greatly intensified; then, when vomiting occurs, as it almost always shortly does, the mind of the attending doctor turns at once to meningitis, especially as he sees the pulse and temperature rising without any change in the condition of the lungs to account for it. Delirium and convulsions follow, and the patient in a very few days becomes comatose and dies.

My neighbour, Dr. Appleton, has kindly provided me with notes of a case which may, in its principal features, be considered as typical of the course of a meningitis following pneumonia:

Mrs. A., twenty-six years of age, six months pregnant with her first child, suffered from pneumonia of the left lung from about October 3 to 10, 1909, the disease terminating by crisis on the latter date. The patient progressed, to all appearance, to convalescence, with the exception of a persistently troublesome cough, but this also gradually yielded to the usual remedies.

On October 22 she complained of slight rheumatic

pains in her limbs. At 1 p.m. on the 26th she expressed herself as feeling much better, dressed, and came downstairs. At 11.30 p.m. on the same day she complained of intense pain, localized to the middle of the cranium, which had come on suddenly at tea-time, and there was much retching, but no vomiting. About half an hour later the doctor was sent for, and on arrival found his patient in a state of violent delirium, throwing herself about the bed and not recognizing anyone; but this condition was somewhat relieved by a hypodermic injection of hyoscine hydrobromate.

On October 27 Dr. Appleton was called at 6 a.m., and found Mrs. A. as bad as ever, but able to take nourishment. The temperature was 101° F.; the urine had a specific gravity of 1020, but contained neither albumin nor sugar; the skin was hyperæsthetic, and the great restlessness precluded any idea of ophthalmoscopic examination; there was no rigidity of the cervical muscles or nystagmus; the pupils were slightly dilated, and reacted to light rather sluggishly. Towards midday the patient complained of intense headache, and also of great pain about the body and between the shoulders.

On October 28, 9.30 a.m., she was much worse, but conscious. At 9.30 p.m. lumbar puncture was carried out by Dr. Cameron, and about 1½ ounces of cerebrospinal fluid withdrawn, which was turbid. Catheterization was necessary night and morning; the bowels were constipated, and not moved by soap-and-water enemata.

October 29, patient much worse, temperature 107° F., and comatose; died at midnight.

Bacteriological examination of the fluid revealed the

presence of pneumococci, which confirmed the diagnosis of pneumococcal cerebro-spinal meningitis.

Infective Endocarditis.—Acute meningitis occurring in those who suffer from valvular disease of the heart is a rare but well-recognized fact. It is seen when there is no other primary disease, and quite as often when there is the double complication of pneumonia and endocarditis.[1] Given a case presenting meningeal symptoms in a person with recognized valvular disease, who also has developed a drenching and persistent sweating, the diagnosis of the double complication of septic endocarditis and acute meningitis may be considered complete. This form of meningitis is always fatal, and post-mortem examination shows both sloughing vegetations on the cardiac valves and a diffuse purulent meningitis, chiefly at the vertex.

Scarlet Fever.—The following case is presented as being characteristic of acute meningitis when it occurs during the course of an acute exanthem :

On June 10, 1896, I saw E. S., aged four years, whom I had previously known as a bright, healthy, and intelligent-looking child, and found her suffering from a well-marked attack of scarlet fever. The temperature was febrile, the pulse a little accelerated, and the tonsils inflamed; otherwise there was at that time nothing unusual in the case.

On June 14, the rash being still in evidence, the mother called my attention to the fact that the child complained much of her head, which seemed to be

[1] *Vide* case reported by Livingstone and Jubb, *Lancet*, p. 486, February 25, 1905.

very hot; and she also asked me if there was anything in the medicine (a diaphoretic) that was likely to be affecting her child's hearing, as she seemed somewhat deaf. I found her temperature to be 103° F.—that is, two degrees higher than the day before, and I thought that the altered condition might be due to a greater intensity of the scarlet fever.

On June 15 it had become quite obvious that the little patient was suffering from cerebral meningitis. She turned from the light, her pupils were moderately dilated, and she had been very restless during the night, constantly raising her hands to her head. There was a great want of intelligence in her face when I asked her how she was. The abdomen was retracted, but there was no retraction of the head. In the evening the pupils were still more dilated, but unequally, and were quite insensitive to light.

On the 16th the pulse was 140, and the temperature 104° F.; the pupils were both widely dilated, and the patient seemed blind. She had had several convulsions during the night; her right arm was paralyzed, and there was also paralysis of the external rectus of the right eye. She was now in a state of stupor, had the dorsal decubitus, and no longer avoided the light.

On the 17th the right leg was also paralyzed; she passed fæces involuntarily, and was semi-comatose.

On the 18th she was deeply comatose, and on the 19th she died.

The facts which most forcibly impressed me at the time were that the temperature was higher, and that the child passed into a stupose and then comatose state

more rapidly than in any case of tubercular meningitis I had ever seen.

The presence of right hemiplegia and left strabismus indicated that the inflammatory process had been intense upon the left motor area and base of the brain (forming the variety *b* in our definition of this disease), and that there was a thick exudate upon, or suppurating foci in, those portions of the cerebral cortex—points which only a post-mortem examination could have settled.

Hemiplegia, without any meningeal symptoms having been observed, has been repeatedly noticed after scarlet fever. Abnormality of the reflexes does not suggest to us that meningitis is either present or imminent, but that either the attack will be a severe one or that some complication—probably cardiac—has arisen.

Measles.—Measles is recognized as a cause of acute meningitis, but the author has had no case, nor has he heard of any in the medical circles in which he has moved. At a meeting of the Clinical Society early in 1887 Dr. Sydney Phillips spoke of a case of purulent cerebro-spinal meningitis supervening on measles, in which the necropsy proved the accuracy of the diagnosis.

Such symptoms as fretfulness, delirium, and stupor, which point to a cerebral origin, may appear, but they pass away in a day or two; they are due to the febrile condition, and perhaps to the absorption of the toxins of the disease. The central nervous system may be deeply affected by measles; acute myelitis has occurred, the patient recovering.[1]

Influenza.—There is much divergence of opinion as

[1] *Vide British Medical Journal*, October 16, 1909.

to the frequency of influenzal meningitis. Some writers say that it produces the largest, and others hold that it supplies the smallest, number of cases of all the acute febrile diseases. Out of 55,000 cases of influenza which occurred in the German Army in a given time, it was only complicated with meningitis on four occasions; yet in the epidemic of 1890-91 one of my near neighbours, the late Dr. Close, had such a case in his practice, and many cases, then and since, have been recorded in the English medical papers. Influenzal meningitis may be cerebral only *in situ*, and serous only in character, producing a mere moistening of the meninges; or it may be purulent and diffuse over the meninges of the whole cerebro-spinal axis.

Before we turn to the consideration of influenzal meningitis, we must be certain that our diagnosis of the primary disease has been correct.

It goes without saying that our patient will have been febrile, complained of headache, had pains in his arms or legs, and had much coryza. All these symptoms, with or without bronchitis, are equally indicative of influenza and an ordinary cold; but if the headache was severe, and we have heard much of pain at the back of the eyes, and noticed a sago-like eruption on the lips and palate, we may be pretty sure that our conclusion has not been faulty.

However alluring it would be to study the general effect of the influenza neuro-toxins on the nervous system—whether it may be neuralgia; neuritis in its many forms; the depression of spirits which so often follows influenza, passing at times into the deepest melancholy or the most violent maniacal insanity;

paralysis or paresis—cerebral, spinal, or peripheral—we must perforce defer the pleasure of dealing with any of these matters at the present time. Possibly we may approach them on some future occasion: much still remains to be said of them.

All who practised in 1889-90, and since, know well that the various psychoses and neuroses are unfortunately real enough in post-influenzal patients. Some of us may, ourselves in the background, have noticed the particular walk or the hesitating speech that betokens grave nervous disorders, or have had anxious parents bring to us their young children suffering from paralysis of the legs, or of one leg and arm, which, except for the history of a recent attack of influenza, we should have to inform them was the frequently crippling disease known to us as acute anterior poliomyelitis.

But we also know, and have seen, neuroses, pretended or simulated by those who have had influenza, who desire a further period of idleness and leisure. At present we must confine ourselves to the question of determining what are the symptoms, during or after an attack of influenza, which indicate cerebral or cerebro-spinal meningitis.

The micro-organism upon which influenza is said to depend, Pfeiffer's bacillus, or one closely allied to it, is usually found in the exudate or cerebro-spinal fluid; but pneumococci, staphylococci, and streptococci are also found.

The late Dr. Close kindly gave me some information very shortly before his death as to the symptoms and post-mortem conditions exhibited in his case. The ill-

ness began with tonsillitis and the usual symptoms of influenza, at a time when influenza was raging in this city. In a few days severe headache was developed; drowsiness soon followed, and passed quickly into deep coma. The usual indications of cerebral meningitis were not noticed, and he depended for his diagnosis upon the intense headache, which was not accompanied by vomiting, and the rapid oncoming of drowsiness. There was no retraction of the head or paralysis of any kind. Post mortem, extensive congestion and œdema of the pia mater were found over both hemispheres, especially the left and at the base, passing into the Sylvian fissures; there was much serum, and lympho-pus.

The phenomena of influenzal meningitis may appear at the onset or height of the disease, or may not show themselves until the patient has been ill some time or begun to recover, although they are most likely when coryza or lachrymation is most severe. The patient may at first only have complained of sore throat, and you find slight or severe tonsillitis; the headache, which may at first have been general or frontal, becomes localized to the vertex or occiput, and the head usually becomes slowly retracted, sometimes to an extreme degree, and the greater the retraction the greater is the probability that the cervical spinal meninges are also affected. Drowsiness and somnolence are characteristic symptoms, and they are those which we least wish to see—indeed, the somnolence may be so great as to simulate death. Vomiting is almost always present, and when present is particularly troublesome; deafness

is not uncommon, and aural giddiness, from probable effusion into one or more of the semicircular canals, may be complained of. The optic discs may be swollen, and if the patient lives long enough, distinct optic neuritis may be present; but in influenza optic neuritis or retinitis may be present without any meningitis. The pupils, which are dilated, become sluggish in their action to light and accommodation, and they may become quite stable and both reactions be lost. Hemiplegia or a paresis of the muscles of the limbs, either of one side or of the lower limbs, is not uncommon, and paralyses of the third, fourth, and sixth cranial nerves are also seen. There may be no convulsions, but choreiform movements of the hands have been noticed by many; there is occasionally a sudden gush of fluid from one or other nostril, which is sometimes followed by a quick subsidence of meningeal symptoms. More frequently this occurs too late to be of benefit, and if such discharge does not take place, pus may be found post mortem in the frontal or other nasal sinus. Kernig's sign, tâche cérébrale, and hypersensitiveness of the skin, may be present.

Mumps. — Cases of acute meningitis, in which the cerebral symptoms were slight and recovery rapid, have been known to complicate mumps.

Uræmic Nephritis.—Chronic nephritis supplies a few cases. The diagnosis, based during life upon symptoms, has been confirmed post mortem by the intense redness and thickness of the cerebral meninges and much congestion and œdema of the grey matter, parenchymatous nephritis being the only assignable

cause. The cortex may be affected without the meninges, and when Broca's convolution is involved, aphasia, with or without right-sided mono- or hemiplegia, follows. Recovery is possible in either case.

Rheumatic Fever.—Acute meningitis may immediately follow—as we have said may be the case with pneumonia—seeming recovery from rheumatic fever. Without assignable reason, headache and vomiting come on; there is a sudden rise of temperature, but not often severe head retraction, and the disease is inclined to run a very rapid course, perhaps within a couple of days of the onset ending in coma and death. The pathology nearly approaches that of post-basic meningitis, in that the inflammation of the meninges and the exudate may be confined to the posterior part of the base of the brain, that the *Diplococcus intracellularis* may be in the cerebro-spinal fluid, and that there may be a general spinal meningitis.

Smallpox.—The author has seen few cases of smallpox during his professional life, and of those few none developed acute meningitis; but he understands from those who have seen much of this disease that variolous meningitis is a fatal, but fortunately a rare, complication. Dr. Hermann, in Nothnagel's 'Encyclopædia,' says: 'To the gross anatomical lesions in the region of the brain belongs, in the first place, purulent meningitis, the relations of which to smallpox are probably metastatic. It is fortunately a rare complication of variola, seems to occur most frequently in children, and is specially connected with the period of suppura-

tion and desiccation . . . there may also be localized encephalitis.'

Typhoid Fever.—In addition to the difficulty there may be at the onset to differentiate typhoid fever from tubercular meningitis, acute meningitis may arise during the progress of typhoid fever. It is suggested by increased headache, rigors, retracted head, increase of the headache, Kernig's sign, etc. Optic neuritis may be present without meningitis. The condition known as 'typhoid spine,' in which, during convalescence, a patient complains of pain on rising, walking, or making any movement in which the spine takes part, and associated with abnormality of the knee-jerks, may be due to a slight spinal meningitis; but it is more often ascribed to acute inflammation of the vertebral periosteum and spinal ligaments. In these cases the *Bacillus typhosus* has been found in the cancellous tissue of the vertebræ.

Diagnosis.—The diagnosis depends very much upon the same symptoms as tubercular meningitis, but the onset is sudden or in connection with some acute disease, and the symptoms develop quicker. Their differential diagnosis will be considered later.

Prognosis.—The prognosis is best in the purulent meningitis of otitis media, whether cerebral only or cerebro-spinal, when removal of carious or necrosed bone, and subsequently thorough drainage, have been carried out. In influenza the outlook is highly unfavourable, especially in very young children; but although many patients die, influenzal meningitis is not so hopeless as the meningitis of other acute febrile

diseases. Cases that recover usually show some improvement on the third or fourth day, drowsiness and somnolence being suggestive of a fatal issue. The purulent cerebro-spinal meningitis of influenza is not necessarily fatal; improvement and recovery have been recorded after from 2 drachms to 2 fluid ounces, according to age, of purulent matter has been removed by lumbar puncture. Recovery or death depends much upon the nature of the toxic agent: a benign meningeal lesion only may result from the toxins secreted by the influenzal microbes; while the graver lesions caused by streptococci, staphylococci, or other such bacilli, cause fatal results. The meningitis of pneumonia, scarlet fever, measles, smallpox, rheumatic fever, uræmic nephritis, and infective endocarditis, has a uniformly fatal ending.

'Neither convulsions, coma, fever to 104° F., unconsciousness, hemiplegia, irregularity of the pupils, nor relaxed sphincters, may cause death. The gravest condition appears to be complete coma, general relaxation of limbs, rising temperature, and Cheyne-Stokes breathing' (Saundby).

Treatment.—Treatment is to be considered under the two heads of ameliorating suffering and an endeavour to cure the disease. For the **first**, quietude, ice-cap to the head, and ice to suck in case of vomiting, with one or other of the analgesics for the headache, may be found useful; subcutaneous injections of the hydrobromate of hyoscine or other sedative may subdue the restlessness, morphine only being used when the symptoms are more spinal than cerebral. For the

second, treatment differs according as to whether the meningitis is of otitic or other origin, the advantage in treating the former being that the *fons et origo mali* can be removed by operation. In otitic meningitis it goes without saying that the tympanum, if perforated, has been incised while the inflammation was yet confined to the middle ear. On the appearance of meningeal symptoms, the full mastoid operation will suffice if the otitis media is recent; while if it is chronic, the complete radical operation will be required, fistulous openings in the semicircular canals, particularly the horizontal, and fistulous tracts in the external labyrinth, being sought for and drained. If the patient's condition has not been relieved by the above measures, it would be correct to lessen the intracranial pressure by the withdrawal of cerebro-spinal fluid. If the spinal tap is dry through the blocking of the foramen of Magendie, a decompressive operation, with incision of the meninges, is justifiable.

In other forms of acute meningitis the most scientific treatment would be the withdrawal of 15 to 20 c.c. of cerebro-spinal fluid, from which a serum should be prepared and reinjected into the spinal subarachnoid space with as little delay as possible; and, medicinally, on the first appearance of meningitis, to administer by the mouth 30 grains of urotropine daily in case of an adult, as this drug has been found, in a case in which the spine was afterwards trephined, to be present in the subdural space one and a half hours after ingestion. In purely idiopathic cases the withdrawal of cerebro-spinal fluid only is necessary, but it should be repeated

if the procedure has in any way improved the patient's condition.

Méningisme.

Méningisme, or pseudo-meningitis, are terms given to those meningeal symptoms which are not founded upon pathological changes in the meninges. There may be a slight hyperæmia of the meninges, but the symptoms are usually dependent upon the effect of toxins on the brain, and completely disappear if the cause of their presence can be found and removed. A similar or somewhat slighter condition often ushers in pneumonia and typhoid fever, and in the course of a day or two generally disappears.

The symptoms, often slight, are those of meningeal irritation, and include most of those spoken of as being present in tubercular meningitis, such as headache, bending back of the head, Kernig's sign, drowsiness, or even coma, hurried pulse, retraction of the abdomen, irregular or immobile pupils, and opisthotonos has been noticed.

Probably those cases of meningitis which are, and remain, serous, and in which the condition never becomes serious, as occurs frequently during middle-ear disease, would at one time have been considered as examples of méningisme; on the other hand, some of the so-called recoveries from tubercular meningitis would perhaps have been more correctly described under this name. If a toxæmic origin is not apparent, the condition may have been caused by the irritation of ascarides or other intestinal parasites.

The diagnosis may be impossible without the assistance of an examination of the blood and cyto-diagnosis, and is then more of a negative than positive character. There may be a lymphocytosis of the cerebro-spinal fluid or not, but there are no polymorphs, and the blood contains an excess of eosinophile cells; the fluid is clear, and flows uniformly and without evidence of intracranial pressure; there is only a trace of albumin, and the reducing power with Fehling's solution is normal; no micro-organisms are found, and no bacterial growth produced in culture media.

Traumatic Meningitis.

PACHYMENINGITIS (*παχύς*, thick; *μήνιγξ*, a membrane) is an inflammation which affects the dura only. The dura mater is a thick and dense membrane, inelastic in children, but becoming more elastic with age. It lines the interior of the skull, adhering most closely at the vertex, and hangs as a loose sheath (the **theca**) in the spinal canal, and consists of two layers, endosteal and meningeal, which are imperfectly separated by lacunæ and bloodvessels. The meningeal layer, by its reduplications, which project into the cavity of the skull, forms the falx cerebri, falx cerebelli, tentorium cerebelli, and diaphragma sellæ, and it is prolonged to the outer surface of the skull through the foramina of the base.

External pachymeningitis, a relatively common disease, is an inflammation of the endosteal layer; and internal pachymeningitis, a rare disease, is an inflammation of the meningeal layer.

Causes.—External pachymeningitis is consecutive to **osteitis** consequent to injury of the skull, such as a kick, fall, or blow, coming into violent contact with any obstacle in the dark, diving into too shallow water and striking the head violently at the bottom, or having the skull pierced by any narrow or pointed weapon (if the latter reaches the brain, encephalitis and probably brain abscess may also result), or to the following diseases: pyæmia, influenza, tuberculosis, syphilis, erysipelas, suppuration in the middle ear, orbit, the nose and its accessory cavities. It may occur as acute or chronic, the former having a septic or tubercular origin, and the latter mostly being due to tertiary syphilis. Incidentally it may be noted that the dura mater may be thickened primarily by deposition in it of syphilitic gummatous material, and the skull becomes diseased afterwards. The inner table of the skull, having undergone severe inflammation, from which it has been unable to recover, on account of there being no outlet for inflammatory products through the blocking up of the diploë, etc., loses its vitality and becomes eroded. Sometimes the erosion extends through the whole thickness of the skull; the dura mater is then affected secondarily, and becomes thickened in patches from cell-proliferation of the epithelial layer, and adherent to the skull, the patches consisting of hard fibrinous material, in which numerous thin-walled capillaries may be developed or gummata be deposited. Pus forms, and is diffused over the surface of the brain, or remains circumscribed from the gluing together of the dura and bone; and the closer the union of these is,

the longer the inflammatory process continues, or the meninges become adherent the one to the other, and they, perhaps, to the brain substance. The micro-organisms in the pus may penetrate into the soft meninges and cause leptomeningitis, or into the nearest cerebral sinus, usually the superior longitudinal, and produce pyæmic sinus thrombosis.

Symptoms. — Pachymeningitis has no symptoms peculiar to itself. The author remembers chloroforming many years ago a patient who had a considerable portion of her skull removed by the trephine for complete erosion, but, so far as he can remember, there were no symptoms other than headache, vomiting, and a general out-of-health state. If the forehead is the part injured, pressure of displaced bone, or, the result of inflammation, pus or serum on the frontal lobe, may cause the development of mental symptoms, or even acute mania, curable only by replacement of displaced bone or evacuation of inflammatory product.[1]

Tumours, benign and malignant, and of the latter endothelioma chiefly, arise from the dura mater, and produce those localizing signs which disease of the subjacent brain would produce. If a person receives a serious injury to the skull, which may or may not be of sufficient force to fracture the external table of the skull or to bring about hæmorrhage into the membranes, or is the subject of any of the diseases enumerated, osteitis may be started, and continue for a week or ten days before any symptoms are observed.

[1] *Vide* 'Mania from Traumatic Meningitis,' *Lancet*, June 25, 1904, p. 1794.

When they do appear they are vague and indefinite: occipital headache and vomiting are almost certain; then may follow deafness, giddiness, and various paralyses; and if they continue unrelieved, he becomes drowsy, then stupose, and soon dies. Rigors are to be particularly dreaded, as they are probably suggestive of pyæmia, which has ensued from particles, detached from gangrenous diploic veins, entering the circulation. When the disease has become thoroughly developed, there may be optic neuritis, and the kind of paralysis present, if any, will depend upon the site of the lesion. Syphilis has a preference for the cerebellar fossa, and tuberculosis the same for the temporal bone.

In pachymeningitis of the cerebellar fossa the symptoms may be suggestive of cerebellar abscess only. In one such case[1] there were deafness, facial paralysis, paralysis and atrophy of right sterno-mastoid, trapezius, and of the right half of the tongue, and paralysis of the right vocal cord. In a case[2] in which no cause, either in the shape of injury or disease, could be assigned, the symptoms were frequent convulsions at the beginning, but no loss of consciousness, and a progressive hemiplegia. The right half of the skull was found coarsely porous, the right half of the dura thickened, and the cortex shrunken and atrophied.

Internal Pachymeningitis.—Internal pachymeningitis is rarely met with, but occurs as 'hæmorrhagic internal pachymeningitis,' or 'hæmatoma of the dura mater,' due to extravasation from the thin-walled

[1] Reported in *Lancet*, vol. i., p. 186, 1892.

[2] *British Medical Journal*, vol. i., p. 710, 1889.

vessels in the fibrinous exudation. Of this variety we have an example in a case[1] in which, four weeks after a fall from a height on to the ear, the patient became giddy, but complained of neither headache nor vomiting. He developed paralysis in the left arm, leg, and facial nerve, had retracted abdomen, and soon became unconscious. Post mortem pachymeningitis was found over the right hemisphere, and on the dura being opened over the right hemisphere the subdural space was found full of blood-clot. This variety also occurs as **purulent internal pachymeningitis.** In a recorded case,[2] there was headache and occasional vomiting, febrile temperature, great irritability, and head drawn down to the left side. Two days before death there appeared right facial paralysis, ankle-clonus, Babinski's and Kernig's signs. Post mortem, there was found a subdural abscess at the vertex of the brain, the pia-arachnoid being natural. All the patients exhibited the Cheyne-Stokes type of breathing some time before death.

Prognosis.—The prognosis is good if the necrosed bone or inflammatory products are removed before rigors occur; bad in the extreme if they have already occurred, as they suggest that infective material has penetrated the cerebral sinuses and been carried into the circulation, and produced pyæmic abscesses in the lungs or other organs.

Treatment.—Treat the causal disease, if it can be ascertained, remove necrosed or carious bone, and

[1] *Lancet*, vol. i., p. 861, 1905. [2] *Ibid.*

evacuate any collection of pus that is found circumscribed.

Arachnitis.—This variety of traumatic meningitis results from the same causes as pachymeningitis, but most often from penetrating wounds. The symptoms are incontinence of urine and fæces, incomplete hemiplegia and hemianæsthesia, noisy delirium, and possibly unilateral sweating. Cold lotions should be applied to the head as soon after an injury as possible; but if the above symptoms become marked, only a decompression operation will be of any value.

Leptomeningitis. — Leptomeningitis results from laceration or puncture of the visceral arachnoid, which rests on the brain, and particularly after fracture of the petrous portion of the temporal bone.

Anatomical Changes.—If the trauma is in connection with the hemispheres, the exuded sero-lymph covers the convolutions and dips into the sulci; if the injury is fracture of the base of the skull, lymph may only be found on the base and ventral surface of the medulla oblongata.

Symptoms.—We may suspect basal leptomeningitis if after an accident there has been bleeding from the ears and facial paralysis, followed by drowsiness, stupor, or other cerebral phenomena. The outlook is bad. The only possible treatment is the persistent use of mercury.

Syphilitic Meningitis.

Syphilitic disease of the cerebral meninges, usually associated with endarteritis of the arteries, and due

to acquired disease in early manhood, has been noticed before the primary sore has healed, but is usually a complication of the late secondary stage, as the osteitis, so often accountable for pachymeningitis, is of the tertiary. It may be acute or chronic, and more often the latter than the former; and the site *par excellence* is at the base and in the posterior fossa of the skull, but the vertex is also frequently affected.

There is a gummatous meningitis in which the soft meninges are mostly affected, but vascular changes in the brain are almost always associated with, and are likely to result in, thrombosis and neurotic softening of the contiguous brain, or hæmorrhage into the brain or its meninges.

Pathology.—The pia-arachnoid loses its translucency, becomes opaque, and in places takes on a dead-white colour; a peculiar gummy substance is found as an exudation, soft as jelly, and greyish-red or grey in colour, which glues together the pia and dura mater, and the former often to the surface of the brain. The tendency of this exudation, sometimes as much as one-third of an inch in thickness, when not interfered with by suitable treatment, is to become fibrinous or honey-comb in character, holding gummata in its meshes or cells, and it powerfully compresses the brain, into which the gummata grow, and such cranial nerves as it may surround. The dura is also at times thickened.

The **intima** of the arteries of the pia-arachnoid and the arterioles of the brain are usually inflamed and thickened, and the external coat has been found in the same state.

Both sets of vessels may be thrombosed, the thrombus showing, by its pale colour, stratified structure and hardness, that it has been formed slowly; and the surrounding brain will be found in a state of yellow or 'necrotic' softening, or the brain substance may be atrophied or sclerosed.

The gumma is a firm, dry, yellow, caseous material, and distinguished from tubercle by the fact that it is irregular in outline, while the latter is almost spherical.

Symptoms.—The symptoms are generally those of the other forms of meningitis, as severe headache—often complained of at the occiput, and worse at night—vomiting, pyrexia, a quick pulse, and optic neuritis, to which mental symptoms are often added. In children, convulsions, with always a considerable retraction of the head and rigidity of the nuchal muscles, often associated with well-marked sixth and seventh nerve paralysis, are common, and, instead of the facial paralysis, there may be only a twitching of the muscles. The symptoms may simulate a cortical lesion.

In adults and older children a staggering or ataxic walk may be observed, due to the presence of the meningeal exudation upon the surface of the cerebellum; and the same occurring in the fourth ventricle and over the medulla accounts for the cranial nerve paralyses and the occlusion of the entrance to the spinal canal, and consequent internal hydrocephalus. In adults, hemiplegia, or monoplegia of one leg or arm, with or without sensory disturbances in them, is common; or the patient may complain that he can only use a leg or

an arm awkwardly, or has a prickling sensation or numbness in that limb; and recovery from a hemiplegia may coincide with the commencement of general paralysis of the insane.

Aphasia also is common, and is as a rule slight, the patient being unable to remember a familiar word, or he finds that he cannot think of or utter an appropriate word. Happily the aphasic condition proves of a transitory nature, and passes away under specific treatment; but however slight and passing it may be, it has been known to forecast imbecility, and in children idiocy or imbecility.

Diagnosis.—The most reliable guides are severe and persisting headache, which is particularly characteristic when it occurs at night-time; and the irregular way the cranial nerves are affected is a striking peculiarity of the disease, nerves whose external origins are distant from each other being most often in fault, and the fact of their being affected early in the disease. Syphilitic osteitis and large gummata are excluded by the early stage of syphilis in which they arise. In cases in which there is doubt as to whether the meningitis is syphilitic, the agency of the microscope will most probably reveal the presence in the blood of the *Spirochæta pallida*—spirally twisted threads discovered by Schaudinn and Hoffman to exist in syphilitic persons—if the case is syphilitic. If the microscopic examination is sterile, the serum diagnosis of syphilis, introduced or modified by Wassermann, should be had recourse to; but the process is complicated, and can only be carried out by those who have access to a laboratory, or persons expert

in laboratory methods.[1] There are considerable differences of opinion as to the value of this test: some observers say that it only gives a positive reaction in 50 per cent. of cases which are known to be syphilitic, while others say more than 90 per cent. of such cases give a positive reaction. The value of the test is on the same lines as the finding or not of the tubercle bacillus in the cerebro-spinal fluid. If the reaction after the Wassermann serum test is positive, the patient is beyond doubt syphilitic; and if it is negative, syphilis is by no means contra-indicated.

Prognosis.—If the true nature of the disease is diagnosed early, and antisyphilitic remedies administered in large doses, the prognosis is good, perhaps better than in any other form of meningitis, and the diagnosis receives partial confirmation in the rapid improvement under mercury; but the gluing of the meninges to the bone and cerebellum and the development of internal hydocephalus are ultimately fatal. Chronic meningeal syphilis may produce no signs, often only an increasing lack of intelligence and drowsiness, and the nature of the disease may only be ascertained post mortem.

Treatment.—The treatment is that of syphilis, with which all readers are doubtless well acquainted. The author only desires to say on the subject that, personally, in cases of this kind he should give a preference to the inunction of mercury in cases of children, and intramuscular injections of the same drug in adults.

[1] *Vide Lancet*, February 13, 1909, p. 481; *La Biologie Médicale*, No. 1, 6e année, Janvier, 1908, pp. 25, 26, 27, 28 *et seq.*

Toxæmic Meningitis.

Toxæmic, amicrobial, or narcotic meningitis is observed most frequently as chronic alcoholic meningitis in those who have been long of intemperate habits. It is an acute toxæmia of the brain, and simulates, rather than is, an actual meningitis; and however characteristically the symptoms may point to the brain or its meninges, the post-mortem appearances of these structures are negative; but there may be a little serous moistening of the convolutions, which has given to the condition the name of 'wet brain.' It may also be observed in lead-workers and those who have subjected themselves to the opium, chloral, or cocaine habit, and in those who have taken large doses of camphor.

Symptoms.—The symptoms in alcoholic meningitis may follow closely upon an attack of delirium tremens. Two or three degrees of fever and a quick pulse present themselves; there is some twitching of the muscles and great hyperæsthesia of the skin; slight optic neuritis may be present, and pain is produced by pressure upon the muscles of the limbs or abdomen.

The tâche cérébrale is easily produced, the pupils are contracted and do not respond to light, the tongue is coated, and the bowels constipated. There may be some retraction of the head and rigidity of the limbs and abdominal muscles.

If the case is to result in recovery, there must be signs of improvement before the oncoming of a very rapid pulse, cold sweats, cold extremities, and semi-coma.

Prognosis.—The mortality is heavy, and prognosis is bad if there is retraction of the neck, and with this stiffness of the limbs and coma.

Treatment.—Nourishing food and stimulants, with only partial withdrawal of the latter if the origin of the illness is alcoholic, constitute the treatment most likely to benefit.

In a child[1] who developed symptoms of meningitis after taking a large dose of camphor the injection of caffeine was followed by recovery.

The **saturnine** form of toxæmic cerebral meningitis is acute or subacute and chronic or latent. In the former variety all the symptoms seen in meningitis may be present, except Kernig's sign; but they are usually transitory, and, as pointing to the plumbic origin, there might be vomiting and diarrhœa, and such nervous symptoms as convulsions, delirium, and epilepsy. In the chronic form, if a dull, heavy, and persistent headache and restlessness occur in a lead-worker, or even in one who is not directly exposed to any of the several sources of lead-poisoning; if he has a sweet odour of breath and a blue line on his gums, or, without the two last symptoms, there occur in an afebrile lead-worker a pronounced anæmic cachexia, colic, and peripheral neuritis, the latter revealing itself probably by a bilateral wrist and foot drop, saturnine meningo-encephalitis may be looked upon as the trouble, the diagnosis being strengthened if there is a lymphocytosis in the cerebro-spinal fluid. The treatment is that proper for lead-poisoning. In lead-poison-

[1] *Vide Lancet*, December 18, 1905, p. 472.

ing, other than meningitis, the author has found analgesics to distinctly benefit the headache.

Posterior Basic Meningitis.

It was only in 1878 that Drs. Barlow and Lee differentiated posterior basic meningitis, under the title of 'cervical opisthotonos of infants,' or, as it might now be called, infantile sporadic cerebro-spinal meningitis, as an ailment distinct, in many of its symptoms and pathology, from tubercular meningitis; and now, through the light thrown upon its study by bacteriological science, mainly by the researches of Dr. Still, we have to acknowledge its close relationship to epidemic cerebro-spinal meningitis, in that it is caused by the same or an essentially similar micro-organism. If we look upon them as two varieties of the same disease, it must be said that they differ widely in their clinical characters, considerably in their pathology so far as the site of the part affected is concerned, but not at all in their actual pathology—the infiltration of the membranes, perivascular infiltration of vessels in the cerebral substance, being the same in the two conditions—and they only differ slightly in their bacteriology.

Pathology.—The pia-arachnoid is inflamed over the posterior portion of the base of the brain—that is, there is congestion and thickening of it from the optic chiasma in front to the pons Varolii, medulla, and adjacent surfaces of the cerebellum behind, never reaching the anterior part of the base or the vertex, but sometimes including that covering the cervical portion of the cord; and in only having this circumscribed area

affected, post-basic meningitis differs from the epidemic disease, in which the arachnoidal disease changes are general throughout its whole extent. There is also much distension of the lateral ventricles, from the filling up of the fourth ventricle with thick gelatinous lymph, or the closure of the foramen of Magendie by adhesions; but there are no characteristic changes in the coats of the arteries, as in tubercular meningitis. The exudation, composed of red cells, leucocytes, and abundant fibrin, is similar to that of the epidemic disease, and agrees with it also in this particular—that in recent cases there is much soft gelatinous material, rather than pus or fibrino-pus.

Cytology and Bacteriology.—The cerebro-spinal fluid is under considerable pressure, is clear in a few and cloudy in most cases, and seldom purulent. In lumbar puncture no fluid may flow—a dry tap—from the reasons already given, and polymorphonuclear leucocytes are almost always in excess of mononuclears. Dr. Still in 1898 examined the micro-organism present in the exudate of post-basic meningitis, and found it to be very similar to Weichselbaum's diplococcus, inasmuch as it also closely resembled the gonococcus, was Gram-negative—that is, did not stain when subjected to Gram's method of staining—was for the most part devoid of capsule, intracellular, and occurred free in the exudation and cerebro-spinal fluid and cells; but that it differed from Weichselbaum's in growing well in agar and gelatine, and had much greater powers of endurance, he being able to keep it in full vitality for three weeks—a period which Dr. Thursfield has since increased to

thirty-eight days—whereas Weichselbaum's did not grow at all, or only very slightly, in these media. Since Dr. Still's pronouncement many observers have found the two micro-organisms indistinguishable, but that they do differ in their culture and agglutinative power. Further, the post-basic micro-organism does not agglutinize or opsonize with a patient's own serum or that of an epidemic case; and in the matter of phagocytosis, the micro-organisms of this disease are not eaten up by the polymorphonuclear leucocytes of the blood of an epidemic case. While these differences are insufficient to constitute two distinct diseases, the micro-organisms seem to have different qualities—differing strains of the same organism, as they occur in epidemic or sporadic cases.

Symptoms.—Post-basic meningitis is a disease of infancy or early childhood. Barlow and Lee stated eleven months as the limit of age, but cases have been seen up to two years, and it is most common during winter and spring. The symptoms are apt to show themselves **suddenly** in the form of vomiting, convulsions, or **screaming**, which may be almost continuous. Extreme **retraction of the head**, suggestively pointing out that the meninges of the posterior part of the base of the brain or cervical portion of the cord, or both of them, are involved in the disease, and associated with rigidity—tonic contraction—of the cervical muscles, often gradually passing into opisthotonos, forms one of the most striking characteristics of the disease. It may be noticed at the very beginning of the illness, or be deferred for days or even weeks, or may only be in evidence towards the close. Optic neuritis is rare, but

some papillitis, with retinal hæmorrhages, has been noticed. On the other hand, **amaurosis**, not depending on any change in the optic discs, is common. A point noticed by many, and said to be peculiar to this disease, is extreme retraction of one or both upper eyelids, so that the child seems to seek the light rather than to avoid it, and the staring look of obviously sightless eyes obsesses one as soon as noticed; but this retraction is not a constant feature of the disease or of the case, as it may never be present at all, or it may be very noticeable for a time, and then for a time the eyes are seen to be imperfectly closed.

Nystagmus is present in about one-half of the cases. Another cardinal symptom, fit to be classed with the head retraction, is **progressive emaciation**, which appears to be an essential part of the disease; it is not due to the vomiting, as it may be present when the latter is quite absent. The pulse is as a rule quick from the first, but it may be irregular and very slow; the temperature is usually below 100° F., but it may be high, and is almost always erratic. In the early days of the disease the respirations are normal, but later they are hurried, perhaps rhythmically so, and they may become of the Cheyne-Stokes character. Early in the disease the pupils are contracted and equal, but later they become dilated. The only cranial nerve likely to be affected is the sixth, and an internal squint is not uncommon. Kernig's sign can almost always be obtained, and often at an early period of the disease; but, like the head retraction, it may be a late symptom. A sucking movement of the lips is also a noticeable

feature. The abdomen may be distended, flaccid, or retracted, and the tâche cérébrale may not easily be obtainable. As in other forms of meningitis, there are restlessness, irritability when moved—probably because all movement increases pain—drowsiness, and afterwards stupor, eventually passing into coma. All symptoms, except drowsiness and an erratic temperature, may be latent until late in the disease. Bulging of the anterior fontanelle is a frequent and early symptom. During the illness there are often attacks of general or localized convulsions; if the latter, they may vary in character, from rigidity of an arm or leg, or both, to clonic spasm, as trismus. The duration of the illness is within wide limits—anything, indeed, from a minimum of five weeks to several months.

I lately attended a child, eight months old, whose symptoms coincided in every particular with those given above; but as it died considerably within what is said to be the minimum time, and there was no post-mortem examination to support or negative my opinion, I see no benefit to accrue from supplying details.

Diagnosis.—Given a child under a year old, with considerable head retraction, whose illness came on suddenly, and whose body is not boat-shaped, the probability is that the illness is post-basic meningitis; but it must be remembered that there may be much head retraction in otitis media, but an examination of the tympanic membrane will remove any difficulty.

Prognosis.—The outlook is not very hopeful, but certainly better than in tubercular or acute meningitis. In cases of recovery the general condition improves and

the temperature lowers before vomiting or head retraction gives in.

Treatment.—Intrathecal injection of Rupell's serum, after the withdrawal of some cerebro-spinal fluid, has quickly removed the worst symptoms and produced eventual recovery. When this method of medication is beneficial, but not curative, post mortem the exudation is found to be excessive over the medulla.

Sporadic Cerebro-Spinal Meningitis.

Our first knowledge of the sporadic (adult) disease slightly antedates that of the epidemic, the third Munro having described the former in 1827.

All that may be said with regard to the clinical manifestations of epidemic cerebro-spinal meningitis is true of adult sporadic cases, even to the division into 'fulminant' or 'malignant' and 'ordinary' cases; but the majority belong to the former rather than the latter class.

Pathology.—The pathology also conforms to that of the epidemic disease, in that the cerebral meninges, particularly the pia mater, are deeply congested, the latter being dull and filmy; the cerebral veins and ventricles are distended, but there is thickening of the meninges when the illness is of less than two days' duration, and there is an exudation of yellowish-green fibrinous pus more or less over the whole surface of the brain and in the sulci of the convexity and base, and along the course of bloodvessels in both situations; or the exudation may be only post-basic, extending as far forward as the optic chiasma, or be most marked there;

and in cases where there is this intense congestion of the cerebral meninges and distension of ventricles there may be no exudate, even if the whole length of the spinal subarachnoid space be filled with blood; or the inflammatory process may be found to have been much more virulent in relation to the cord than the brain, as there may be no exudate deposited by the cerebral meninges, and the fluid in the ventricles be serous, yet there may be a purulent pachymeningitis, internal and external, running the whole length of the cord. In addition, there is that peculiar gelatinous material in the subdural space of the spinal meninges and at the base of the brain which has been found both in the epidemic disease and posterior basic meningitis.

The lungs may be congested or collapsed; the solid organs take part in the widespread inflammatory condition, the spleen being sometimes soft and diffluent; the liver exhibits the same yellow mottling on the surface, and there may be the same hæmorrhagic pleuritis as in the epidemic disease.

Cytology and Bacteriology. — The cerebro-spinal fluid may be clear, turbid, or purulent, and it may flow uniformly or in spurts. There is an excessive leucocytosis, mostly polymorphonuclears, but also some mononuclear cells.

It is only when we come to the bacteriology of the disease that we arrive at a dividing-line of a sort, which reveals the typical *Diplococcus intracellularis meningitidis* of Weichselbaum only, found lying in groups of from one to four pairs of cocci; or—and more frequently—the capsulated **extra**-cellular diplococcus—

otherwise the pneumococcus—only, although the patient may not have suffered from pneumonia; and often the bacillus of influenza or the *Bacillus typhosus*, even if the patient has never suffered from influenza or typhoid fever. If both the meningococci and pneumococci are present, the latter are likely to be in greater numbers.

Symptoms.—The sudden commencement, often with a rigor or convulsion (or the patient is found unconscious at his work, in the street, or in bed); the intense headache and vomiting; the great pain down the centre of the back or in the abdomen; the bleeding from the nose; the extreme retraction of the head, which is thrown back between the shoulders; the cervical rigidity, with or without the head retraction; the restlessness and stupor; the lateral decubitus and knees drawn up; the erratic up-and-down temperature—now febrile, then normal or subnormal, in all cases, or hyperpyretic in fulminant ones; the rapid and feeble pulse; the presence of Kernig's sign in the early stages of the disease, and its liability to be absent later; absence of the reflexes, the corneal in particular; marked hypersensitiveness to touch and sound; and sometimes herpes of the lips and neck, erythematous and petechial rashes, and the dusky mottling of the skin, so remindful of typhus fever; delirium and coma, may all be met with in sporadic cases, and only the *fons et origo mali* may differ slightly.

As a set-off against this similarity of symptoms, the disease in an ordinary case runs a milder course than in the epidemic type. There is often also in the ordinary cases some degree of optic neuritis, and there is **usually** an absence of the herpes and rashes so characteristic of

the epidemic disease. Individual cases have shown a spasmodic state of the muscles of the lower limbs, with painful twitchings, incontinence of urine and fæces, great tenderness of the calves of the legs and of the feet, photophobia, epistaxis, and choreiform movements of the arms and legs.

'This disease, in patients other than infants, is sometimes marked by exacerbations approaching the malarial type, often of daily occurrence, the pulse and temperature rise, frontal or vertical headache, stiffness of neck and vomiting, with dulness and drowsiness, may come on and remain for twelve or more hours, and then remit, and during the succeeding twelve hours the patient is in an apparently comfortable state.'[1]

When we think we have before us a case of sporadic cerebro-spinal meningitis, it must not be forgotten that similar symptoms may be produced by totally different agencies than the micro-organisms mentioned, as in the extension of a purulent cerebral meningitis due to influenza or otitis media.

Diagnosis.—A disease of sudden onset, with headache and vomiting, pain down the middle of the back, great head retraction, and an irregular temperature, is probably the sporadic disease, but only lumbar puncture, with examination of the fluid, can make the diagnosis conclusive.

Prognosis and Treatment.—Prognosis and treatment of the sporadic disease must be on similar lines as for the epidemic, but it may be stated that the mere

[1] Clinical Society's Transactions, vol. xxxviii.

removal of purulent cerebro-spinal fluid has ended in recovery.

The two following cases will illustrate the difficulty one may have, relying upon clinical symptoms only, in saying to which variety of cerebral meningitis any particular case may belong.

On May 25, 1894, I was asked to see a boy, aged five and a half years, who had been out of sorts several weeks and ill for a week. He had no aural discharge, nor had he had any recent exanthem or other acute febrile disorder. On looking at my patient, the most obvious symptom presented to me was the extreme retraction of the head, causing me instinctively to think of cervical caries—cerebro-spinal meningitis at that date did not occupy the professional mind to anything like the extent it has done in recent years. On examination of the spine, I found no tenderness or pain, but the cervical muscles could be felt as rigid bands. To the mother the most important feature of her boy's illness was the headache, which was referred to the forehead, and which, always present, became at times almost intolerable, causing him to scream out loudly, his screams being heard some distance from the house; the paroxysms occurred at intervals of a few hours, day and night, but were worse at night, and during them he would place his hands on the side of his head, as if the pressure afforded relief. The temperature was 100° F., and was never higher than 101° F. during the illness; the pulse, from the onset to convalescence, was quick. The tâche cérébrale was easily elicited, and the abdomen was boat-shaped. The illness began with a two days'

vomiting, which recurred at intervals. He laid upon his left side with the knees drawn up and feet pointed, was rather deaf and avoided the light, his pupils were dilated, and a week after I first saw him distinct optic neuritis was detected. There was no ocular or other paralysis at any time, nor did convulsions ever occur. The breath was not particularly offensive, but the bowels only acted after enemata, and became still more obstinate during convalescence, and he urinated into the bed-clothes.

No family history of syphilis or malignant disease was available, but several relations, on both father's and mother's sides, had died of consumption. There was no albumin in the urine, no red spots on the abdomen or back, nor were the rashes of spotted fever even observed. The headaches were of such a distressing character that the mother expressed herself to the effect that she would be satisfied if they could be relieved, whether recovery took place or not. The case went on for weeks with little improvement or change; sometimes the patient was delirious, but there was never stupor nor coma. The arms were frequently, but not always, flexed; the thighs were flexed and toes pointed.

On June 30 the temperature was normal, headaches were less frequent, and retraction of the head less marked; the patient was less irritable—irritability and fretfulness having been previously pronounced symptoms —than formerly, and he no longer avoided the light. On July 8 there was no headache nor retraction of the head, and he could again bear himself upon his feet.

As regards the reflexes I have no notes. My former patient is now a sturdy young man, but for some years after his illness he was subject to occasional severe headaches lasting a day or two.

At the time my impression of the nature of the illness was that it was an acute meningitis, which I might now qualify by saying that it had not passed out of the serous stage, and probably localized to the posterior occipital fossa, although I was not then familiar with the term 'posterior basic meningitis'; indeed, I believe the term had not then been coined, but now, when the case is recalled to me, on seeing the young man at his home or in the street, I have grave doubts as to the exactness of my diagnosis. If I say the case was one of cerebral meningitis, I may be on safe ground; but if I go farther and give it a more definite title and say tubercular, simple idiopathic, or sporadic cerebro-spinal meningitis, I may be wrong.

The history of tubercular disease on both sides of his parentage, the invariably slight pyrexia, and the insidiousness of onset, may entitle me to say that it was the first-named disorder; while the severity of the headache, the extreme retraction of the head, and absence of rashes, point forcibly to the second; the intense headache, extreme retraction of head, long illness *plus* recovery, point to the third; while the method of onset, the moderate and regular pyrexia, the absence of rashes and joint troubles, were against it. But if one can rely upon the strongly pronounced opinion of Professor Osler, and it would be difficult to name a higher authority, it was in all probability the last named. Dr.

Osler says: 'Given a case with all the well-marked symptoms of meningitis, recovery is a strong warrant that we had to deal with the form due to the *Diplococcus intracellularis*.'

Last summer I had an opportunity of seeing on several occasions a somewhat similar case in the Sculcoates Union Infirmary, in the absence of Dr. H. Robinson, Medical Officer of the Institution, by whose courtesy I am able to refer to it.

A young man, twenty-two years of age, was brought in by the police with the information that he had had a fit in the street. The man was in a dazed state and remained so for some time, frequently complaining of pain in his head. He lay on his left side, shunning the light, and his head was drawn back until it formed a right angle with his back.

He had no joint pains, spots, or rashes, and no further convulsion, but was at times delirious, and he eventually fell into a stupose state, and remained so for many weeks, and then gradually recovered.

He was sent to the seaside to assist him to regain his strength, but he lost flesh; later he developed a cough, and tubercle bacilli were found in copious quantity in his expectoration.

The probable sudden onset of the illness, extreme head retraction, and long illness *plus* the eventual recovery, are all strongly in favour of sporadic cerebro-spinal meningitis; but the fact that the lungs shortly afterwards gave evidence of tuberculosis almost leads us to think that the brain trouble was tubercular meningitis, protracted to an unusual length, which will,

after a longer or shorter intermission, reappear, if the lung trouble does not in the meantime end fatally.[1] At any rate, the immediate development of the lung trouble raises doubts as to the variety of meningitis which did not exist before.

Epidemic Cerebro-Spinal Meningitis.

Perhaps no disease has received more attention during the last quinquennium than epidemic cerebro-spinal meningitis, alike in the matter of its clinical manifestations, bacteriology, pathology, and treatment. We may later assign reasons why we should or should not couple with it cases of (adult) sporadic cerebro-spinal meningitis and infantile sporadic cases, or posterior basic meningitis—the many epidemics and sporadic cases which have occurred in this country and abroad having afforded opportunities in which these questions have been studied. It is only comparatively recently that the disease has become known to medicine, the earliest outbreak in the British Isles having occurred so lately as 1830, and elsewhere not previous to the nineteenth century.

All are agreed that epidemic cerebro-spinal meningitis or fever, or, as it is sometimes called, 'spotted fever,' depends chiefly upon one micro-organism, the *Diplococcus intracellularis meningitidis*, or, shortly, meningococcus, which was first isolated and described by Weichselbaum in 1887, and which is found in the exudate upon the base of the brain, in the cerebro-spinal fluid, the blood, and in the secretions of the nostrils and

[1] Died of hæmoptysis, August 22, 1910.

accessory nasal cavities. Intensity marks the disease throughout its course, alike in the degree of inflammation of the structures and organs affected as in the symptoms exhibited.

Ætiology.—It is more frequent in males than females, occurs more in the class of people who live under insanitary conditions than in the well housed, in those who are exhausted from overwork or athletic exercise, occurs epidemically, and is infectious but considered only slightly contagious; but its contagiousness cannot be ignored, seeing that several persons in the same house are affected at the same time, forming what is known as 'the multiple invasion' of houses, and the first person to become affected is the one who has been nursing in the same disease elsewhere.

Adolescence is the period of life in which this disease most commonly occurs, 75 per cent. being before fifteen years of age; but persons of all ages are liable to attack, from infancy upwards. The season of the year has a predisposing influence, as it is certainly most frequent in winter and spring. The incubation period is from three to four days.

Pathology and Morbid Anatomy.—It has been said earlier in this work that there are few cases of acute cerebral meningitis which are not associated with more or less inflammation of the brain itself; and the remark applies with equal force to the epidemic disease—so much so, indeed, that 'meningo-encephalitis' would again represent the condition more truly than 'meningitis.' Generally speaking, the disease begins with intense hyperæmia of the meninges of the brain and

cord, causing a fibrino-purulent, sometimes gelatinous, exudation; the inflammation spreads to the grey substance, and the ventricles fill with a sero-fibrinous or purulent fluid. In places the encephalitis may break down, and form small foci of greenish-yellow pus; but the condition of the brain and membranes differs according to the type of the case. In the **fulminant, foudroyant,** or **malignant** type, in which death may ensue within three or four hours, or well within thirty-six hours, after the first appearance of symptoms, the cerebro-spinal fluid is not much increased, and may look like pure blood. There is always a condition of acute congestion of the head and soft meninges, and also of the surface of the brain, the small vessels exhibiting marked engorgement. There is rarely thickening of membranes or exudation, but copious exudation has been seen in cases which have died within a few hours; and in those who have lived a few days it has been sufficiently thick on the first day to cause hemiplegia and aphasia. The cut brain appears as if infiltrated with blood-clot, and in some cases the subdural space is filled with gelatinous material. When death takes place after a two-day's illness, the cerebral dura mater shows intense congestion, and the vessels of the pia mater are also infected; the membranes are often glued together in places and to the surface of the brain, wherever there may be patches of encephalitis. The brain substance is hyperæmic, red, and softened, and a thin, yellowish fibrino-purulent exudate is found more or less over the whole of the cerebral surface, particularly in the interpeduncular space, over the pons Varolii,

and upon the under surface of the cerebellum, and less copiously over the convexity and sides and along the cord; or it may only occur in well-defined localized areas. In these cases and those which live longer there may be a purulent infiltration of the neurilemma of some of the cranial nerves—most often of the sixth, less often of the third and seventh. In those cases in which the headache is chiefly **vertical**, fibrino-pus, or a yellow opalescent serous fluid, is found chiefly filling up the sulci on the upper surfaces of the hemisphere, and in less quantity over the base, but most over the medulla oblongata. In the **post-basic** cases the exudate is found chiefly confined to the posterior part of the base of the brain and the medulla, and for varying distances down the cord; and pus has been observed altogether outside the spinal dura. When death occurs after the patient has been ill some weeks, the pia arachnoid is much injected, thickened, and very adherent, and the whole of the hemispheres are covered with a fibrino-purulent fluid and much lymph, the latter sometimes being as thick as jelly in the larger sulci and fissures; the exudate is yellow or greenish-yellow in appearance, and is often more fibrinous than purulent, especially at the base. The lateral ventricles are usually filled with a turbid fluid. The cerebro-spinal fluid is always greatly increased in volume, and consequently, under great pressure, has an increased quantity of albumin and little or no reducing agent, is often turbid, but sometimes clear, only being bloody in the fulminant cases; it soon becomes purulent, pus lying in the meshes of or underneath the pia arachnoid. Adhesions may occur

between the spinal meninges, and the cord, particularly the cervical portion, is hyperæmic, and at times is in a state of myelitis as far down as the lower dorsal region. If the disease resolves and the patient recovers, the exudate may undergo complete resolution through disintegration of pus cells and absorption of the fluid portion, or it may become semi-organized; and this, with the pia arachnoid remaining permanently thickened, may cause the development of mental symptoms at a future time; the fluid in the ventricles becomes absorbed, or it remains as chronic internal hydrocephalus, whether the foramen of Magendie is closed or not.

The morbid anatomy of the disease goes far beyond the brain and spinal cord; even in twelve hours there may be intense congestion of many organs and structures. It is not uncommon during a patient's life to be aware that he is suffering from hæmorrhagic nephritis, acute bronchitis, congested or collapsed lungs, pleural hæmorrhages, acute suppurative pericarditis, acute suppurative arthritis of the larger joints, often of the knee; and post mortem there may be found pleural adhesions, congestion of the suprarenal glands, hæmorrhagic congestion of the mucous membrane of the jejuno-ileum and duodenum, and the solitary glands in the latter, but seldom the Peyer's patches of the former. The glands at the angle of the jaw may be enlarged, as also are those of the mesentery, and generally there is a great addition of lymphatic tissue; the thymus may be enlarged, and there may be a mottling of the surface of the liver, due to irregularly situated yellow patches,

probably of fatty degeneration. The spleen does not often participate in the general congestion, but it may be softened, and purpuric patches on the skin may occur after death when there has been none during life.

Cytology and Bacteriology.—The blood in fulminant cases is dark, even tarry in appearance, and very liquid. In most cases it shows a high polymorphonuclear leucocytosis, usually 30,000 to 36,000 per centimetre, but it may be as low as 12,000 or as high as 60,000; but such a low count is not suggestive of the disease. 'A low count of leucocytes means that the patient can offer only a feeble resistance, and a high count a profound invasion;'[1] on the other hand, a relatively high count may be met with in tubercular meningitis and some general diseases. The absence from the blood of eosinophile cells (cells which readily become stained with the rose-red dye eosin) in the early part of the illness signifies the present serious condition of the patient, or that he will be ill a long time. The cerebro-spinal fluid also has an excess of polymorphonuclear leucocytes, and some neutrophile cells, both of which are obtainable by centrifugalization, but may be deposited in standing, the supernatant liquid being rejected and the deposit examined microscopically.

Epidemic cerebro-spinal meningitis depends mainly on the irritation caused by the *Diplococcus intracellularis meningitidis* of Weichselbaum, but other micro-organisms, chiefly the extracellular diplococcus, may be present. Weichselbaum described the micro-organism, which he considered the causative agent, as diplococci

[1] Larkin, *Practitioner*, June, 1909.

of varying size, always occurring in pairs or tetrads, occasionally elongated, typically flattened on their opposed surfaces, so that each coccus appeared to be of semicircular form resembling gonococci in appearance, in that they were Gram-negative and lay within the cytoplasm of the leucocyte. He also found the pneumococcus in one-fourth of his cases, but considered them as of only secondary importance. Flexner has later found, and it is a point of far-reaching character, in differentiating them from similar organisms, that they only retain their vitality a few days and grow very badly in media. Later observers have found with one or both of these diplococci the *Staphylococcus albus*, the *Bacillus enteritidis* of Gaertner, the tubercle bacillus, streptococci, the anthrax bacillus, slender cylindrical, immotile rods. The typhoid bacillus, or *Bacillus typhosus*, short rods 1 mm. to 3 mm. in length, by 0·5 mm. to 0·8 mm. in diameter, with rounded ends, and often growing into long threads, has been the only organism found in some cases, even when the patient has not had typhoid fever recently, or where there are no typical typhoid lesions in the intestines. True chains of the intracellular cocci do not occur, but two or three pairs may form irregular rows.

The organism rapidly disappears on isolation, through the destructive agency of a chemical ferment or enzyme, which is liberated on the death of the cell; but since the earlier discoveries of the qualities and powers of the meningococcus it has been found that, when preserved in sera media at 37° C., and prevented from drying, strains are viable up to 100 days, while cold or sus-

pension in salt solution is very injurious to it. As the pneumococcus has been found present without the meningococcus, opinion is divided as to which micro-organism is the specific one, one side supporting Weichselbaum, and that the pneumococcus is only a degenerative variety of the meningococcus, the other side holding the opposite view.

The site and properties of the meningococcus are that it is found in the exudate on the surface of the brain, both at the vertex and base, in the cerebro-spinal fluid, the serum of the blood, and in the mucus of the nostrils and its accessory cavities. It is Gram-negative and is agglutinized and opsonized to a marked degree by serum from other cases of cerebro-spinal meningitis. Further research may possibly indicate differences, both in cells and bacilli, between fulminant and ordinary cases. If the meningococcus is not found at the first or second examination, it must not be concluded that it is not present, as it has been found after repeated examinations have been negative.

In the Belfast epidemic the post-basic meningitic micro-organism (Still's coccus) was not opsonized or agglutinated by serum from cases of epidemic cerebro-spinal fever, and was thus separated as a distinct variety, or species, from that described by Weichselbaum. Better results are obtained from culture examination than by the direct method, blood culture giving the poorest results. In the later stages of an epidemic it may not be possible to isolate the meningococcus, as it may have died, or, having undergone great changes, it may resist culture.

Classification.—Cerebro-spinal meningitis may be **primary**, when it includes the epidemic, sporadic (adult), and (infantile) posterior basic meningitis; or **secondary**, due to infections of various kinds, as from tubercle, otitis media, and the micro-organisms of those diseases mentioned under Acute Meningitis. The usual method of classifying the **primary** is into—

(1) **Fulminant, Foudroyant, Malignant**, or **Apoplectic**, cases, associated with profound toxæmia and rapidly fatal; and

(2) **Ordinary**, those cases showing symptoms typical only of ordinary meningitis; or into—

(*a*) **Vertical**, in which the purulent exudate is chiefly confined to the vertex of the brain, and

(*b*) **Post-basic**, in which the exudate covers the medulla and extends for varying distances down the cord. Severe frontal headache is characteristic of the vertical, and marked head retraction of the post-basic, but the rule is not absolute.

Symptoms.—Onset of startling suddenness, severe headache referred to the back of the head and neck or forehead, vomiting, pains down the centre of the back, great restlessness, extreme retraction of the head, and a rash, are peculiar to all epidemics of this disease—excepting some of the fulminant cases, which may begin with sudden unconsciousness—and are the essentials in the way of symptoms; but epidemics vary in details. In one headache is unpleasantly prominent, in another joint troubles, in another the rash, and in another the head retraction may be so severe as to be opisthotonic.

It is always an intensely virulent disease, its virulence

being significantly shown in the fact that a person has died in a few hours from an acute septicæmia caused by the meningococcus, without his exhibiting any meningeal symptoms whatever, or causing any pathological change in the meninges, the only symptom being a profuse hæmorrhagic purpura, with increased number of respirations, and a generally collapsed condition, which quickly passed into coma.

Primary epidemic cerebro-spinal meningitis is characterized above everything else by the suddenness of its commencement; but in a few cases there may be a few days of malaise, headache, pain in the back, and vomiting. If the case belongs to the fulminant or toxæmic class, we may find the patient has suddenly become delirious or semicomatose, having had trifling symptoms previously. If the meningeal symptoms about to be mentioned are very slight, or altogether absent, diagnosis may depend altogether on bacteriological examination, as it may be impossible clinically to distinguish cerebro-spinal fever from other acute toxæmic states, as ptomaine-poisoning, uræmia, or malignant purpura. Of the fulminant cases, the following conditions and sequence of events may be observed: A man is suddenly taken ill at his work, or a woman during the course of her domestic duties, or a child at school or at play. After vomiting perhaps once, he becomes suddenly unconscious, the unconsciousness probably alternating with periods of great restlessness, or the patient may be awakened out of his sleep by headache and pain. When seen shortly afterwards by a doctor, the pulse is very quick, the head may be already retracted, with

much rigidity of the nuchal muscles; his face is pale, livid, or perhaps cyanotic, and with or without red patches here and there; his eyes may be bloodshot, his pupils dilated, or at other times contracted; he breathes rapidly and perhaps stertorously, and as the hours pass the breathing becomes more and more rapid, until in a child it may be 80 per minute, and in an adult 40 to 70. The temperature may be normal or subnormal, but there is more likely to be two or three degrees of fever, and it may gradually increase until it is hyperpyretic; but a low temperature does not indicate a mild attack, nor a high one necessarily a severe attack. Kernig's sign is present throughout, but Babinski's only during the close of life. The conjunctival reflex is only just appreciable, both sphincters are paralyzed, and there may be a transient, erythematous or scarlatiniform, mottling of the skin. The patient may succumb well within thirty-six hours, and rarely lives longer than a week, when the symptoms exhibited are a little less serious, and may include optic neuritis; the illness may close suddenly by crisis on any day up to the tenth. Some patients who have presented symptoms almost toxæmic, and neither die nor end their illness by crisis, may gradually pass into the ordinary type.

If the case belongs from the first to the **ordinary** type, the patient, after perhaps only a few hours of **malaise** and a feeling of chilliness, finds himself suffering from **severe headache**, with pains shooting **down the spine** and into the limbs, and increased when the body is moved, almost always forming a prominent and often distressing feature, which may continue to the end, or

be lost after the first day or two. There is also a noisy, often violent, delirium; convulsions are common at the onset, both in children and adults, and may be general or confined to the muscles of the face. Vomiting, probably of greenish-yellow matter, occurs in the majority of cases, but may be only on one occasion. It comes on suddenly, and is often associated with rigors in adults or convulsions in children; and when there is no vomiting, there may be nausea, and the tongue as a rule is dry and cracked, or coated with white fur. There may be a distressing thirst, and the bowels are confined. But in fulminant and some ordinary cases there is loss of control both over bowels and bladder, with bedsores as a consequence in the latter variety. The patient **continually cries out**, if unrelieved by sedatives; but the cry is not the hydrocephalic cry, except in infants, and he is extremely restless. The temperature very often is high, or it may be low at the beginning; but in either case it always runs a markedly **irregular** course, as in malarial fevers. The pulse, always feeble, may be quick, at first it is often slow, but certainly quickens as the disease progresses. The position in bed is for the most part lateral, with the legs flexed. Rigidity of the nuchal muscles, with or without head retraction, is constant, and observed almost at once, whenever there is any attempt at flexion or turning of the head. In point of time, this condition comes after headache and vomiting, but is present in a greater number of cases. It is volitional, as it disappears during deep coma, and when for any reason it becomes necessary to administer an anæsthetic, as in lumbar puncture in young children.

Within a few days—it may be within twenty-four hours of the first appearance of symptoms, often on the fifth day, but occasionally deferred until late in the illness—**retraction of the head**, one of the greatest and most obviously distinguishing marks of the disease, shows itself; the back may become more and more concave until it is markedly opisthotonic, making deglutition impossible. In an extreme degree the latter condition only occurs in the acute stage, and is unusual then, or there may be a rigidity of the whole muscular system. Kernig's sign is said to be always present in adults, and mostly present throughout the illness in children, but it may be absent at a late stage, and there also may be noticed early a rigid contracture of the hamstring muscles, which is particularly obvious when the patient is raised to a sitting posture, and the patellar reflex cannot be elicited. Babinski's sign of the extensor plantar reflex is seen very seldom. There is nothing constant regarding the tendon reflexes, but for the most part they are lost early. The knee-jerk was lost in the first two or three days in twelve out of fourteen cases; the abdominal reflex is absent in about one-half the cases; it may disappear as toxæmia more and more possesses the patient. The corneal reflex is lost always after the disease has become fully developed—indeed, **conjunctival anæsthesia** is justly considered as one of the principal objective signs of the disease. The pupils are usually dilated from the first, are equal and react to light; they may be unequal; they may occasionally be contracted at first, and afterwards become dilated. Mydriasis is far more commonly seen than the opposite

condition. Late in the disease they may again become contracted; they usually continue dilated, but become inactive to light. Photophobia is common in the early stage, and lateral nystagmus is said to occur in one-half of all cases. Optic neuritis is uncommon, but may be present, and conjunctivitis is often seen in children. Hemiplegia has been noticed within a few hours of the onset of the illness in fulminant cases, and so has aphasia, and paresis of one or more limbs is common in ordinary cases. Of cranial nerve paralysis, the sixth nerve is almost the only one to be affected, and thus convergent squint is often seen. Ptosis and facial paralysis are also seen. Diplopia may be present, but disappears as recovery progresses.

The breathing may be normal at first, but in a few days becomes rapid and sighing, and in cases which are to end fatally quickly, it may very rapidly pass into the Cheyne-Stokes type. In some epidemics, inflammation of the joints, especially of the knee, and simulating rheumatic fever, is much in evidence, and may be noticed before cerebral symptoms are exhibited; in other epidemics, arthritis is scarcely ever seen. Abdominal pain is at times very troublesome, and may have a nervous origin; but when associated with diarrhœa, it depends upon those intestinal pathological states already spoken of. It often introduces the patient first to the notice of the doctor, in whose mind the possibility of ptomaine-poisoning may be raised, and later there may be tenderness of the abdomen on palpation. **Herpes labialis**, which may become hæmorrhagic and afterwards scabbed, particularly on the right side of the

mouth, and extending sometimes to the nose and neck, is scarcely ever absent, and is looked upon as a favourable sign; but it may not make its appearance until the end of the third week. The chin is also an occasional *locale* of herpes. Next in importance to the headache and retraction of the head comes the specific **rash**, which is seen in all epidemics amongst white people and in many cases; but it may not occur in negroes, and may be difficult to make out in dark-skinned people. The spots may come out in crops, disappear, and again reappear; some may disappear on pressure by the finger, reappearing when the pressure is removed, as in erythema; or they may be unaffected by pressure, as in petechia, the latter form occurring more frequently than the former. The rash appears about the fifth day, or a little later, the individual spots varying in size from a pin's head to a walnut, or more frequently about half the size of the latter, the larger being obviously formed by the coalescence of many of the smaller. They are sometimes elevated; all seem hæmorrhagic—probably from effusion of dissolved hæmatin into the true skin—and are crimson or dark purple in colour, particularly the larger ones. In some epidemics they are situated mostly on the chest and upper abdomen, in others only on the dorsa of the feet, and this is the special site in fulminant cases; but the arms and legs may be also more or less covered, or the rash may only be seen on the back or buttocks.

The opinion, founded on the similitude of the rash, that typhus fever and epidemic cerebro-spinal fever was one and the same disease, was held by many

physicians in pre-bacteriological days, when the microscope played a less important rôle in the investigation of disease than it does to-day. The late Dr. Murchison held that the two diseases were identical, chiefly because of the fatal character of epidemic cerebro-spinal meningitis, and its being frequently associated with purpuric rashes; others controverted this opinion by pointing out that typhus was seldom seen in children, and was rarely fatal to them, while the other disease was often seen in children, and was very fatal to them.

The author is not familiar with the typhus fever rash, and consequently not in a position to offer any opinion on the matter; but the medical officer of a large fever hospital, and presumably well acquainted with both rashes, has personally assured him of their great similarity. Dr. Chalmers, Medical Officer of Health for Glasgow, describes the rash as 'a few spots, petechial in character, few in number, and irregularly distributed . . . more pronounced than a typhus rash'; and later, in describing the rash on a child of seven months old: 'The features of a typhus rash were exactly reproduced within limited areas of the skin—in fact, the rash presented no feature by which it could be distinguished from that of typhus fever; it had the appearance of subcutaneous stippling, which could be obliterated on pressure.' 'The onset of typhus is not so sudden; the temperature is more regular and more apt to disappear by crisis; the face is more congested, leucocytosis is slight or absent, and the urine gives the beautiful diazo-reaction of Ehrlich.[1] This reaction occurs when urine,

[1] *Vide* Larkin, *Practitioner*, June, 1909.

previously alkalinized with ammonia, takes on a deep red colour when treated with a mixture of sodium nitrite, hydrochloric acid, and sulphanilic acid.

Besides herpes of the lips and the distinctive rash, there may be present urticarial wheals.

Next in importance to the rash as a symptom—and, unlike it, a constant one—comes the great and rapid **emaciation**; and, partly from this cause, feebleness continues long, and convalescence is protracted.

The glossy skin, described by Paget, and due to loss of trophic power, may also be noticed upon the dorsa and soles of the feet.

As emaciation progresses, hæmorrhagic blotches, or patches of chloasma, appear in the skin of the flexor surfaces, and they may be looked upon as forecasting a protracted illness.

The urine is acid and clear, may contain albumin, be bloody, and reduce Fehling's solution. Hæmorrhagic nephritis may be present for a few days, and then disappear.

The patient may be deaf on both sides, and remain so. Painful skin sensations are diminished in the early stage, but hyperæsthesia obtains later to such an extent that the patient may cry out if the bed is moved.

He soon assumes a drowsy condition, but is able to answer questions; is irritable, and resents being moved, as if movement increased his headache or backache. Stupor, beginning early and persisting during the illness, and passing in fatal cases into deep coma, is the usual mental state; but in mild cases the mind may be always clear.

The tâche cérébrale may be present, but is more frequently absent or difficult to obtain; nor is retraction of the abdomen a frequent symptom, in both of which particulars this disease differs from tubercular and acute meningitis.

Infection has never been traced to the water or food supply, as in typhoid fever, yet some hold that the alimentary canal may be the method of infection; nor to the infiltration of the subsoil with sewage, as in diphtheria; but all observers are strongly in favour of the direct method from person to person, as people may live together in close association without becoming infected, if there is no personal contact. It must also be markedly infectious, aerial by fomites, as persons living remote from each other, without any possible chance of contact or infection by 'carriers,' are affected about the same time. That epidemic cerebro-spinal meningitis, like typhus fever, is greatly favoured by living in insanitary conditions was well demonstrated in the Lisbon epidemic, by its occurrence in that city amongst the very poor, ill-nourished, out-of-work, and uncleanly, while the well-to-do, living quite near, who were probably more cleanly in their persons and surroundings, did not have the disease. The meningococcus has often been found in the nostrils of those who have attended the sick, but who have not themselves sickened; and this site seems to act as a nursery of the micro-organism, whence it is transmitted to those who have not been in contact. Dr. Flexner takes the opposite view, and says the organisms may be air-borne; but he agrees in the infection being carried by inhalation into the upper air-

passages, and thence carried by the lymphatics to the subdural and subarachnoid spaces of the brain.

In some epidemics this disease has not been characterized naturally by any crisis—in the Glasgow epidemic there were some cases which terminated naturally by crisis during the acute stage—and unless cut short by serum treatment or death, it often means a lengthy illnesss; and while four months may be looked upon as the usual length, ten weeks is quite common, and six or even ten months is not unusual. Other writers say that abrupt natural termination is most infrequent, but that crisis very often occurs after serum treatment; the temperature falls, and all other symptoms suddenly cease, and the patient is practically well.

It seems fully recognized that severity in the type of the disease considerably moderates after an epidemic in a neighbourhood or city has continued from six to twelve months; in other words, the disease has a natural tendency to exhaust itself, as there are fewer fulminant cases, illnesses are not so protracted, and the symptoms in ordinary cases are not so severe.

Complications and Sequelæ.—The complications are many and serious, including epistaxis and hæmorrhage from the bowels; synovitis, especially of the knee, often ending in suppuration; hæmorrhagic nephritis; diarrhœa; parotitis; conjunctivitis; keratitis; iritis; optic neuritis; destructive panophthalmitis in the later stages; and, during epidemics of cerebro-spinal meningitis, pneumonia of low type, without observable nervous symptoms, is frequent.

In mild cases there are no sequelæ; in a few the

patients are rather deaf on recovery from fibrous ankylosis of the ossicles, and a still smaller number are absolutely deaf in both ears to both aerial and bone conduction of sound; loss of memory and even mental impairment have been observed in both natural and serum-treated recoveries in fulminant cases. Blindness may result from opacity of the cornea after keratitis, or from secondary atrophy of the optic disc due to optic neuritis. After fulminant attacks a person may for a long time be subject to headache, especially when stooping, and there may be paralysis of the ocular muscles, or of a leg or arm, and spastic or flaccid. Happily, the beneficent influence of time removes most of these post-meningitic troubles, except the deafness and blindness.

Diagnosis.—Diagnosis is assisted if an epidemic is prevalent in the neighbourhood. Clinically, we depend on the sudden onset, severity of the headache, extreme head retraction, rapid emaciation, the specific rash and joint troubles. In fulminant cases the two first-named are sufficient if they occur when undoubted cases of spotted fever are about.

Prognosis.—A foreknowledge of the probable ending of a case may, to some extent, be gathered from an examination of the cerebro-spinal fluid, in regard to its appearances and chemical and microscopic characters, and also from the clinical conditions observed.

When the leucocytes are altered and stain badly, the outlook is not good. The younger the epidemic, the more numerous the fatal cases; and the older the epidemic is, the more numerous are the recoveries.

After the first week a morning remission of the temperature is favourable, but severe relapses are very likely; the loss of the knee-jerk throughout the illness is unfavourable, and a dry tap on lumbar puncture, through closure of the foramen of Magendie, is highly unfavourable, but not quite hopeless. Internal hydrocephalus, usually due to the same cause that brings about a 'dry tap,' is not always due to that cause, as it may be present when fluid flows after lumbar puncture, and may cause death after bacterial action is less effective, or has ceased. A semi-organized exudate at the base and sides of the brain may cause death or the development of dementia. Aphasia, after apparent recovery, denotes such a copious or thick exudate over Broca's convolution as to invite us to consider the case as a serious one that will probably have a fatal issue. In the same class are those cases in which the specific rash is present, which we may look upon as barometric in significance, giving us surface indications of the occurrence of hæmorrhage in solid internal organs; it may be said that the bigger the individual spots, and more widespread and purple the rash, the more hopeless is the case. Amongst the unfavourable signs, severe early depression and continuous coma must have a prominent place.

Dr. Graham Forbes sums up the prognosis as follows:

Cases which have become Chronic and have the Best Chance of Recovery.—The cerebro-spinal fluid is clear, and there is only a faint trace of albumin; the diplococci are degenerate and few, and only identified by film preparations; cultures are sterile.

Subacute Cases, in which the patient may live on for a few weeks. The fluid is clear or slightly turbid, very little albumin, polymorphonuclears are not in great excess, and there are a few meningococci in film preparations, seldom in culture.

Severe Cases.—Mostly fatal, very few recovering. Fluid turbid, with much albumin, polymorphonuclears in great excess, and the meningococci can be identified both in film and culture preparations.

Treatment.—It is now acknowledged by those who have seen much of epidemic cerebro-spinal meningitis, with a few important dissentients, that the most successful treatment is the injection into the spinal theca of meningococcal serum—subcutaneous and intravenous injections are of much less benefit, and perhaps inert, because the serum is bactericidal and only slightly antitoxic—and it is averred that this treatment reduces the mortality to one-fourth. The great advantage which intrathecal injection possesses over other modes of introduction has been demonstrated by observers treating series of cases in the same epidemic subcutaneously, intravenously, by lumbar puncture *sine* injection of serum, and subdural injection, and invariably obtaining immeasurably better results from the last method, the first being found useless, and the second and third of small benefit. This is just what we might expect to be the case when the curative agent is brought or not brought into direct contact with the meningococcus and its toxins, and the inflamed meninges which generate the micro-organisms.

The serum is prepared from the blood of horses of

known health—monkeys are the best subjects, but their supply is too limited—immunized by inoculation with fresh agar cultures from the meningococcus obtained from typical cases. Alternatively the serum may be obtained from the blood of convalescents or from the patient himself—after a sufficient time (that is, not less than a fortnight) has elapsed for the formation in the system of strong antibodies—by whipping it as soon as it is drawn, and subjecting the defibrinated portion to a long centrifugalization of 3,000 revolutions per minute; and the serum obtained by the latter method often succeeds when that obtained through the horse has proved useless. It should be used early in the disease—the earlier the more beneficent is said to be its action—in doses of 10 to 20 c.c. for children, and 20 to 45 c.c. for adults, always being previously warmed to body heat, after an equal volume of cerebro-spinal fluid has been withdrawn. Sometimes only one or two twenty-four-hourly injections are necessary, when the disease may subside suddenly as if by crisis, and twelve-hourly in fulminant cases, or in any case if, notwithstanding its use, the patient becomes worse; in other cases it must be repeated daily, and less often after signs of improvement. Serum-therapy decidedly shortens the length of the disease—indeed, under its use the duration is said to be only one-fifth of the time when not so treated.

The mortality is highest in infants under one year old—before the introduction of serum treatment all such patients died; now, with intrathecal injections of Flexner and Jobling's serum, one-half of them recover

—and least in children between five and ten years of age, rises gradually, but surely, with the advance of years, until in the elderly it approaches the infantile rate.

When a high temperature falls to normal, or an irregular one loses its up-and-down character, headache is lessened and vomiting ceases, and there is a diminution of coma, delirium, and insomnia within a few hours or days after the first or second intrathecal injection, we are almost bound to look upon the improvement as due to the treatment. Relapses after serum-therapy are as frequent as they are in cases where no serum has been used; but if the relapses are treated on the same lines as the original attack, patients do well.

In addition to the improvement noted in the patient's condition after about four days of serum treatment, an examination of the cerebro-spinal fluid demonstrates that its effects upon the cellular elements and causative micro-organism have been towards the return to normal in the case of the former and their destruction in the latter, the degenerated polymorphonuclears occurring in diminished numbers, and their place being occupied by normal polymorphonuclears; the meningococci are much reduced in numbers, or altogether absent; and, if present, their inoculations on culture media are sterile.

On a rough estimate, intrathecal treatment reduces the mortality from 90 to 30 per cent., or less, and to 18 per cent. in cases in which it is used during the first week of the illness. Dr. Emmett, New York, says that in his cases the mortality was reduced to 25 per cent. where the serum had a fair trial—*i.e.*, in cases not moribund or almost so when it was used; but all writers

concur in the opinion that it is of little value in fulminant cases. Messrs. Flexner and Jobling had 75 recoveries out of 393 cases treated with antimeningococcic serum, including 11 recoveries out of 22 children under one year of age—cases in which the prognosis is usually considered hopeless. These experienced physicians make the following hopeful and definite assertion: 'It is our belief that the analysis of histories of cases of epidemic cerebro-spinal meningitis which have been presented in this article[1] furnish convincing proof that the antimeningococcic serum, when used by the subdural method of injection in suitable doses and at proper intervals, is capable of reducing the period of illness, of preventing in large measure the chronic lesions and types of the infection, of bringing about complete restoration to health in all but a very small number of the recovered, thus lessening the serious deforming and permanent consequences of meningitis, and of greatly diminishing the fatalities due to the disease.'

It has not been found that this treatment is in any way injurious, or that it emphasizes existing symptoms, but serum rashes may follow. If the pus is too thick to flow, some normal saline solution should be injected and then allowed to flow out. This should be repeated again and again, if necessary, until the thickened pus becomes liquefied, and after such procedure the serum may be injected. Although lumbar puncture and the withdrawal of an ounce or two of pus slowly cannot be relied upon as having a curative effect, there is no question as

[1] *Journal of Experimental Medicine*, September, 1908.

to its power of relieving distressing symptoms, and notably the severe headache. The mere removal of fluid or pus must have a twofold power for good: it tends to lessen intracranial pressure and displaces a number of toxic micro-organisms, and to the extent of what has been removed provides an opportunity for the secretion of a healthier and amicrobial fluid.

Drs. Currie and Macgregor,[1] whose experience was gained in the Glasgow epidemic, May, 1906, to May, 1908, give results very different to those of many other writers with regard to serum-therapy in this disease. They mostly used the subcutaneous or intravenous methods, and their largest dose was 25 c.c., instead of from 30 to 45 c.c., and their serum was not Flexner and Jobling's. They say: 'The administration of serum to our cases was followed in individuals by no consistent modifications of the course of the disease.' But they also say: 'The effect of serum would appear to have been to improve by 20 per cent. the prospect of recovery of cases which survived the first ten days of illness.' They also add that perhaps the epidemic in Glasgow was more virulent in type than others.

Up to the present it has not been found that the post-basic meningitic serum has been useful for the epidemic disease, and certainly serum from the latter disease, although often tried, has invariably been found useless in the post-basic disease. On the other hand, we are told of cases in which the blood from patients with the epidemic disease did not agglutinate typical meningococcal cultures, but reacted well with cultures

[1] *Lancet*, October 10, 1908.

from sporadic post-basic cases. Collargol, electrargol, and lysol have also been used intrathecally with much success, but it has not been proved that they are superior to serum. In one case in which collargol was used with happy results, it is worth mentioning that the cerebro-spinal fluid became sterile after it had been in contact with the silver solution six days.

All other treatment is secondary, but by no means useless; ice-caps and analgesics, guarded by stimulants, are useful for the headache and excitement, and bromide of sodium may change restlessness to quietude; but chloral is contra-indicated. Subcutaneous injections of morphia relieve pains in the back and limbs, and for the same purpose leeches may be applied to the back of the head and along the spine. Hot baths, when the temperature is high, afford much relief to the general condition, as well as relief to the headache. If paralysis continues after convalescence is progressing, iodide of potassium will hasten the absorption of the exudate upon which the paralysis probably depends, and iron tonics will generally expedite recovery.

As it has been shown that this disease is propagated by carriers, something ought always to be done by way of prevention, and those in contact with the sick should be directed to use antiseptic mouth-washes and to syringe their nostrils or inhale antiseptics of such a strength as will be likely to destroy the meningococcus without injuring the soft parts.

CHAPTER II

THROMBOSIS OF THE CEREBRAL SINUSES

Definition.—Thrombosis is the formation of a solid mass or plug in the living heart or vessels from the constituents of the blood (Welsh) by coagulation and agglutination. Thrombosis of the cerebral sinuses is said to be (*a*) primary, marantic or marasmic, simple or non-infective, when it ensues or depends upon an enfeebled circulation and the prostration of debilitating diseases; and (*b*) inflammatory, extrinsic, secondary, or infective, when it is produced by the irritation of contiguous necrosed or carious bone, and the passage through the vessel walls of infective material.

Ætiology.—The clotting of circulating blood is a complex process, many causes being at work for its production, and it is not possible to say which is the most important one. The anatomical emplacement and structure of the sinuses and veins, weak circulation, debased state of the blood, formation of fibrin, an increase in the number of platelets, increase of calcium salts, and agglutination, are all factors, each perhaps acting more powerfully on some occasions than others.

No factor in the causation of cerebral sinus thrombosis is more powerful than the one first mentioned, while each primal disease is productive of a cause peculiar to itself, as by impoverished blood in primary anæmia or a deficient supply of it in post-hæmorrhagic anæmia and after an exhausting diarrhœa.

The cerebral sinuses want altogether the middle or muscular coat present in veins, and in place of an external coat they have only the enfolding of the dura mater, the vessel being consequently fixed, and on account of this unyielding state of the walls there is produced in effect a rigid incollapsible tube. There is also their great width, triangular form and the occasional crossing of trabeculæ, and the manner in which they are emptied during inspiration with walls that cannot contract upon a changing current. The manner in which some cerebral veins join the longitudinal sinus provides an additional cause, so far as that sinus is concerned, in that the cerebral veins over the occipital and parietal lobes of the brain join the sinus at an acute angle from behind forwards—that is, against the stream—and so may possibly retard the stream, but cannot accelerate it, and this retarding influence is further assisted by the veins ascending to the sinus.

Fibrin is not a normal constituent of the blood; it is produced by the action upon fibrinogen of a fibrin-ferment, thrombokinase, in the presence of a soluble salt of calcium. Thrombokinase exists normally in certain glands and muscular tissue and in the walls of bloodvessels, is evolved on the destruction of red blood-corpuscles, and is produced when the physiological con-

dition of the blood is interfered with, as in inflammation—on account of the thinness of the outer tissue of veins, inflammation in the neighbourhood passes readily to the endothelium of the intima—or injury to the walls of the vessels, and, in case of infective thrombosis, the introduction of micro-organisms.

Agglutination is supposed to be due to the formation of agglutinin—a substance having the power of gluing together platelets and red corpuscles, and these to the walls of vessels. Platelets are circular, oval discs, found in blood in the proportion of 1 to 8·5 red corpuscles; they were formerly considered to be derived from the corpuscular elements of the blood, but are now looked upon as independent elements, are usually present in increased quantities in thrombosis, but this condition may be present when there is a decrease of platelets. Neither excess of calcium salts nor slowing of the blood-current in themselves can cause thrombosis.

The purely primary or marantic thromboses, which owe their clotting power to a debased condition of the blood only, occur most frequently in anæmia, both primary and post-hæmorrhagic, and chlorosis; next in point of frequency in influenza; then in children, chiefly under six months old, who have suffered from prolonged diarrhœa; sufferers from bronchial diseases, scarlet fever, measles, tuberculosis, and congenital syphilis come next, but much less often; and it occurs with great rarity in pneumonia, typhoid fever, diphtheria, and in people who are apparently well.

That the tendency to thrombosis is far more marked in chlorotic and anæmic girls under twenty years of

age than in others of the same age is beyond doubt, but the reason is not usually clear why the blood should clot. The frequency of thrombosis in chlorosis is more apparent than real, probably because most of the cases which occur are reported in medical literature. Leichtenstern had only 11 cases of thrombosis (not confined to the cerebral sinuses) in 1,653 cases of chlorosis, and out of 86 cases the lower extremities were affected in 48, cerebral sinuses in 29, and no other situation was represented by more than one instance.

The diminution of the number of red corpuscles and their contained hæmoglobin is a powerful, but in itself not a sufficient, cause of thrombosis; nor is the sluggish state of the circulation, nor even its arrest, because when a vessel is doubly ligatured thrombosis does not necessarily follow. The tendency to thrombosis has been ascribed by Erkhurst to changes in the intima of the vessels, while Rocket believes it is due to the increased number of platelets. In chlorosis the diminution of the amount of hæmoglobin is more pronounced than the decrease in the number of red corpuscles: while the latter may be normal in number, the amount of hæmoglobin may sink one-half or more. Probably, therefore, the diminution of hæmoglobin in chlorosis, assisted by the increased number of platelets and white corpuscles, and the slow movement of the latter on the mural side of the blood-stream, plays an all-important part in the chlorotic production of cerebral sinus thrombosis.

In the exhausting diarrhœa of early infancy the blood is diminished of its due quantity of serum, and is therefore lessened in volume in the whole of the circulation,

the vessels of the brain included. The latter may become dilated, and the causal disease having already weakened the heart's action, we have in consequence an interference with the functions of the red corpuscles and a retardation of the circulation, all which make for clotting in the sinuses. In the causation of sinus thrombosis in influenza there is also a marked decrease in hæmoglobin, the destruction of many blood-corpuscles, and an increase in the white blood cells. Thus we have similar causes working for thrombosis as in chlorosis, and what is wanting in the less debased condition of the blood in influenza is partly, perhaps, accounted for by the injurious action of the influenza bacillus.

In pneumonia and typhoid fever thrombosis affects the cerebral venous channels rarely.

In the later stages of pneumonia we see the great liability of the blood to clot in the occasional presence of thrombosis of the femoral vein. Ante-mortem clotting in the heart is also one of our great dreads in our conduct of a case of that serious disease. In 367 cases of pneumonia observed in one of the London hospitals, thrombosis of the cerebral sinuses was only noticed seven times, and of these four were in anæmic girls; so, perhaps, only in three cases was the thrombosis due to the pneumonic state. The presence of carbonic acid in the blood makes it more liable to clot, probably through its destructive action on the red corpuscles and their hæmoglobin, the presence of oxygen being well known to have a retarding influence over coagulation. This theory of causation is supported by the fact that it is in the later stages of pneumonia that thrombosis occurs,

when the blood has become surcharged with carbonic acid; the presence of the pneumococcus is also said to accelerate the clotting process.

In typhoid fever we see the same liability of the blood to clot in the presence occasionally of femoral thrombosis during early convalescence; and as we know there is a great increase in blood-plates in the third and fourth week of typhoid fever, a consequent thrombosis looks very like cause and effect. The cause of blood-clotting in typhoid fever may be due to the large ingestion of milk as diet, milk containing a large quantity of carbonic oxide. Secondary thrombosis includes (*a*) those cases which owe their clotting power to a special marasmus, plus the introduction of septic matter, as erysipelas, septicæmia during the puerperal state, and such septic local conditions as furuncles, abscesses on the face and lips, anthrax in the same situations, general pyæmia, and injury to bone, particularly when there is inflammation of the diploë; (*b*) cases due to the introduction of septic matter only, as in extradural abscess; and (*c*) extrinsic cases.

The cause of cerebral sinus thrombosis occurring during the puerperium is, perhaps, largely due to the considerable amount of fibrinogen which has been stored in the blood, much more than normally exists. The introduction of septic matter in these cases is primarily due to the **lochia** having become foul; then minute particles obtain entrance by the uterine lymph channels into the venous circulation, and septic thrombosis follows. This reasoning applies to the femoral thrombosis of puerperal septicæmia, as well as to that of the cerebral sinuses, but why in the latter it should

always be the cavernous sinuses which are thrombosed is not quite obvious. It is fortunate for the patient if this septic thrombosis is confined to one side, because it almost always means the destruction of the eye behind which it is situated; happily it was so confined in a puerperal case the author saw on one occasion. Erysipelas has also special predilection for one or both of the cavernous sinuses; and besides its septic properties and altered blood-corpuscles, it possesses a special cause in the tendency of the leucocytes to migrate through the walls of vessels.

The causes of extrinsic sinus thrombosis are those acting upon the outside of the vessel, in the shape of irritation by necrosed bone or necrotic brain tissue, the latter being caused by an infective inflammation, plus the introduction to the sinuses of micro-organisms by the vasa vasorum in the walls of the vessel. The most frequent example of irritation by necrosed bone is that of the petrous bone after middle-ear disease; less often by necrosed bone due to punctured or comminuted fractures of the skull, which have become septic by communication with unclean surface wounds; then by necrosis of the orbital plate of the ethmoid in connection with abscess of the ethmoidal cells; and, very rarely by necrosis of the body of the sphenoid in empyema or abscess of its sinus. When necrotic brain substance is the irritant, the usual rule of the larger vessel being first occluded is departed from if the necrosis is not very near the surface, and the blood in the smaller vessels clots first, from which the thrombotic process extends to the larger vessels, where it may stop, or finally into the sinuses. When extrinsic thrombosis

becomes septic, the route of infection is by infective micro-organisms, such as streptococci, staphylococci, and pneumococci, by veins or lymphatics through inflamed bone, which by contiguity of tissue has started inflammation in the wall of the sinus, and so started the thrombotic process. An additional cause of clotting in extrinsic cases is the frequently associated phlebitis, inflamed walls being a powerful impetus to other causes of thrombosis.

The following table shows the divisions of cerebral sinus thrombosis according to the cause, and the sinuses most likely to be affected by the primal disease:

Cases due to a Debased State of the Blood only.

PRIMARY.	Anæmia and chlorosis	The longitudinal sinus most often, occasionally the cavernous, more rarely the lateral and straight sinuses or the veins of Galen.
	Diarrhœa	
	Influenza	
	Scarlet fever	
	Pneumonia	
	Typhoid fever	

Cases due to a Debased State of the Blood plus the Introduction of Septic Matter.

SECONDARY.	*Infective.*	Erysipelas	Cavernous sinus mostly, then the longitudinal or veins of Galen.
		Furuncles, abscess, or anthrax on face and lips	
		Puerperal sepsis	Longitudinal and cavernous.
		Traumatic and inflammatory causes, as depressed fractures and spiculæ of bone	Superior longitudinal.
		Cases due to Introduction of Septic Matter only.	
		Extradural abscess	Lateral sinus mostly.
	Extrinsic.	*Cases due to the Irritation of Necrosed Bone plus Infection.*	
		Petrous portion of temporal bone	Lateral, sigmoid, petrosal, straight.
		Sphenoid and ethmoid bones	Cavernous.
		Basilar process of occipital bone	Lateral.
		Cases due to Necrotic Brain Substance.	
		Longitudinal sinus and cerebral veins usually.	

Pathology.—Primary or marantic thrombosis usually affects the single median or azygos sinus—that is, the superior longitudinal. Essentially the process of blood-clotting is as follows: The circulating blood consists of an axial, or central, and a peripheral, or mural, stream. The leucocytes are carried along the latter—*i.e.*, next to the walls of the vessel—and the red corpuscles and platelets in the centre of the current. When the blood is debased and the current sluggish, the leucocytes, chiefly polymorphonuclears, with some platelets, through the action of agglutinins, become adherent to the walls of the vessel. The normal condition of the blood being now seriously interfered with, fibrin is developed, and adheres to the already formed sticky mass, and red corpuscles in large numbers are caught in its meshes. In extrinsic thrombosis the irritation of necrosed bone produces a phlebitis; or if the phlebitis arises otherwise, there is roughness and irregularity on the internal surface of the intima, platelets in large numbers, along with leucocytes, become agglutinated to the roughened walls, and the process completes as in other forms of thrombosis.

The recent primary clot is red and at first soft, and in a few days becomes dense, firm, and resistant, non-adherent to the walls of the sinus, and decolorized, or at any rate changes from red to a pale grey colour; and if the blood is still circulating, a white colour results; if stagnation has resulted, the red colour continues, and, on account of being formed by successive depositions of fibrin and the corpuscular elements already mentioned, it is laminated. The clot may first form in the tributary vein and be limited to it, or afterwards extend into the

sinus. The thrombus may not quite occlude the lumen of the sinus, or it may be tunnelled, thus enabling the ordinary circulation to be carried on; if the occlusion is complete, the congestion of the adjacent meninges and œdema of the brain are great, the congestion is relieved by the neighbouring tributary veins, if they are patent; but if one or more cerebral veins are also occluded in association with that of the longitudinal sinus, necrosis of brain substance in the affected area must ensue. The tendency of the primary clot is to become absorbed or organized. The organization begins by bloodvessels being projected from the vasa vasorum in the tunica intima into the periphery of the thrombus; a granulation tissue is developed; fissures are formed, into which leucocytes pass; and a more or less organized tissue is the outcome, absorption proceeding *pari passu* with organization. Red softening and œdema of the brain cause the small capillaries to lose their support and rupture, giving rise to numerous small points of hæmorrhage; on the other hand, hæmorrhages may not be present even in anæmia. There is also excess of serous or sero-sanguineous fluid in the ventricles and the subarachnoid space, and if the fluid passes from the latter position into the tissues behind the eye, ophthalmic phlebitis and abscess may result, with consequent proptosis of the eyeball.

Secondary or infective thrombosis affects the dual sinuses and avoids the two extremes of life. The secondary clot is stratified and softer than the primary, but has a less tendency to be stratified, and is more likely to be adherent to the wall of the vessel, which

is frequently inflamed and is very liable to disintegrate. It may be firm, adherent, and aseptic in one part, and septic and broken down in another; disintegration may start either from the centre or either end. Whether the clot first forms in a sinus, or first in a tributary vein and extends to the sinus, lethal micro-organisms pass through the vessel wall from the primary seat of the disease. On account of the infective nature of the inflammation of the walls of the sinus, they become soft and thick, the epithelium is loosened; and the clot becomes puriform, and if there is total occlusion micro-organisms are carried by the reversed current—possible by the absence of valves in the sinus—into the descending blood-stream; or if the disintegration has caused the lumen of the sinus to be renewed and the blood again to circulate, infective matter is swept into the general circulation and deposited in distant organs, usually the lungs, causing at first pulmonary infarction, and later on abscesses, or into the intestines, producing infective enteritis.

The list on pp. 108-110 gives some idea of the lesions that may be found associated with cerebral sinus thrombosis; and it will also be noticed that females, of whom a large proportion are young girls, far outnumber males. Symptoms are only mentioned when they have been severe or appear unusual.

Symptoms.—The subjective symptoms of cerebral sinus thrombosis are the general symptom-complex of acute cerebral leptomeningitis, and include sudden onset, headache—which may be general, or have the pain located at the vertex, temples, behind the ear, or

No.	Sinuses and Veins affected.	Sex.	Age.	Primal Disease.	Associated Lesions and Symptoms.
			Years.		
1	Superior longitudinal and lateral	Male	—	None given	Numerous hæmorrhages on surface of both hemispheres.
2	Superior longitudinal and cerebral veins	Female	25	,,	—
3	Cavernous - - -	,,	31	,,	Left hemiplegia.
4	Lateral - - -	Male	27	,,	Extradural abscess.
5	Cavernous, double and left lateral	Female	32	,,	—
6	Cavernous, double filled with pus, left lateral gangrenous, and left petrosal filled with red thrombus	—	—	Empyema of sphenoidal sinus	—
7	Lateral - - -	,,	14	None given	Mastoid antrum and tympanum filled with granulation tissue.
8	Superior longitudinal and lateral	,,	—	,,	Meninges adherent to the bone.
9	Jugular vein at the bulb -	Male	14	,,	Pus in mastoid cells.
10	Cavernous and lateral -	—	25	—	—
11	Cavernous - - -	Female	19	No cause found	—
12	Superior longitudinal and cerebral veins	,,	16	Chlorosis	Œdematous softening of brain, with bloody extravasations.
13	Superior longitudinal and cerebral veins	,,	—	,,	—
14	Transverse, lateral and great vein of Galen	,,	19	,,	—
15	Straight and large vein of Galen	,,	17	,,	—

16	Longitudinal, right lateral and superior petrosal	Female	13	Anæmia	Heart dilated and hypertrophy of mitral valve; lungs and liver diseased; pain and frequent jactitation of left arm; twitching of mouth; squint.
17	Superior longitudinal, lateral and veins of Galen	,,	25	,,	Great restlessness and tossing about of arms.
18	Veins of Galen - -	,,	20	,,	Dura mater adherent to brain; lateral and straight sinuses partly empty.
19	Longitudinal, lateral and cerebral veins	,,	2	Diarrhœa	Blood in other sinuses dark.
20	Longitudinal - -	,,	26	Anæmia	—
21	Veins of Galen - -	,,	8	Scarlet fever	—
22	Straight and right vein of Galen	,,	13	Post-scarlatinal	Right optic thalamus enlarged and in state of red softening; rigidity of neck, with some head retraction.
23	Straight - - -	,,	—	Measles	Left arm flexed and a little rigid; temperature before death 108° F.
24	Lateral, straight and cerebral veins	,,	30	Puerperal sepsis	Congestion of both optic thalami, and veins of both legs were blocked.
25	Cerebral veins - -	,,	23	,, ,,	Ecchymosis of upper surface of left hemisphere; right-sided convulsions.
26	Longitudinal and cerebral veins	,,	35	,, ,,	Headache very severe; optic neuritis and convulsive movements.
27	Middle cerebral vein -	,,	25	,, ,,	—
28	Lateral, full of foul-smelling pus	Male	66	Influenza	—
29	Veins of Galen and collateral vessels	Female	—	Facial erysipelas	Congestion of the optic thalami and caudate nuclei, with minute hæmorrhages into the floor of lateral ventricles.
30	Cavernous - - -	Male	—	Blebs on lip and eyelid	—

No.	Sinuses and Veins affected.	Sex.	Age.	Primal Disease.	Associated Lesions and Symptoms.
			Years.		
31	Cavernous - - -	Male	—	Pustular swelling on upper lip	—
32	Cavernous, double - -	,,	—	Sore on upper lip	Meninges about cavernous sinuses covered with lymph; orbital tissues on right side inflamed and swollen.
33	Superior longitudinal and cerebral veins	Female	—	Pyosalpinx	—
34	Cavernous - - -	,,	22	Tooth abscess	—
35	Cavernous, double - -	,,	18	,, ,,	Septic basal meningitis.
36	Cavernous and circular, pus and organized clot -	Male	28	Abscess of sphenoidal and ethmoidal cells	Pus on surface of brain.
37	Longitudinal and straight -	Female	4	Otitis media	Pus in left tympanum and mastoid antrum; no necrosed bone.
38	Longitudinal and left lateral and subclavian veins	,,	7	,, ,,	Kidneys shrunken and left heart hypertrophied; great rigidity of nuchal muscles.
39	Lateral, purulent - -	,,	7	,, ,,	Pulmonary infarcts.
40	Cavernous - - -	,,	9	,, ,,	—
41	Lateral - - -	Male	25	,, ,,	—
42	Lateral, contained fœtid pus	,,	9	,, ,,	Pus in mastoid antrum.
43	Lateral - - -	,,	14	,, ,,	Extradural abscess.
44	Lateral - - -	,,	38	,, ,,	—
45	Lateral - - -	Female	9	,, ,,	Thick fibrous band occupied place of jugular vein and vagus nerve.
46	Superior longitudinal, lateral and cerebral veins	,,	—	—	Numerous hæmorrhages on surface of both hemispheres.
47	Superior longitudinal, probably	,,	18	Chlorosis	Recovery; right femoral vein thrombosed; unconscious thirty-eight hours, and then gradually recovered.

on the forehead—quick pulse, hurried respirations, vomiting, general malaise, optic neuritis, drowsiness, irritability, and convulsions—general or unilateral, and often succeeded in the latter case by hemiplegia, both in children and adults, or delirium in adults, the local or objective symptoms differing according to the sinus affected.

Longitudinal Sinus.

The site of thrombosis in purely marantic cases—that is, in weak and debilitated people—is in the single or azygos sinuses, and most frequently in the superior longitudinal sinus, or in the dilatations of the frontal and parietal veins as they enter that sinus, and the veins of Galen; but the latter may be thrombosed in company with any of the sinuses. The characteristic feature of thrombosis of this sinus is œdema of the skin of the vertex and engorgement of the scalp veins and those of the forehead, the last indicating that the anterior portion of the sinus is affected. There is occasionally epistaxis, almost always severe vertical headache, vomiting, drowsiness, often optic neuritis. Children are liable to convulsions, which, when unilateral, bespeak thrombosis of the cerebral veins of one hemisphere, as well as that of the sinus. There may also be pain down the spine, rigidity of the nuchal muscles, and tremor of the lower limbs. If the objective signs are wanting, as they frequently are, upon what are we to depend for our diagnosis?

If a young girl suffering from breathlessness and œdematous lower limbs is obviously anæmic, also com-

plains of severe vertical headache, with double vision due to paralysis of the sixth nerve and failing eyesight due to optic neuritis, thrombosis of this sinus is very likely to be her ailment, although optic neuritis in the anæmic does not of itself suggest thrombosis. When no objective symptoms are exhibited, the patient only presenting cerebral symptoms of an anomalous character, the diagnosis may have to rest upon the fact that the patient is anæmic, and possibly venous thrombosis in other parts, as in the left lower limb. When anæmia is the cause of sinus thrombosis, the veins of Galen are liable to be associated, and may rupture into the lateral ventricle and cause rapidly fatal results. The patient suddenly shrieks and falls; she is found to be unconscious, her pupils are widely dilated and eyes staring, the breathing is stertorous, the sphincters lose their power, the conjunctival reflex disappears, convulsions set in, and death follows in two or three hours.

As regards diarrhœa, we may find the following sequence of events: A child, probably an infant under six months old, has suffered from diarrhœa from a few days to as many weeks, when fits supervene, probably after improvement seems to have set in, and the number and character of the stools have changed for the better. If a convulsion chances to be in progress at the moment of the doctor's visit, he may observe that it is wholly confined to one side. If the child survives the fit, he may be left comatose, and die in two or three hours; or he may live on, and succeeding convulsions may be general, and symptoms very similar to those of meningitis may develop. The anterior fontanelle, if still open,

will be depressed, and the cranial bones may overlap; the scalp over the site of the longitudinal sinus becomes swollen and œdematous, and the veins there and on the forehead stand out distinct and turgid; there may be rigidity of the cervical muscles; the child sleeps much, and eventually becomes comatose, and death probably results. In such a case, in the absence of a post-mortem examination to prove the contrary, we are justified in concluding that there is thrombosis of the longitudinal sinus, and that the clotting began first in the cerebral veins on the side opposite to that on which the first convulsion occurred. Had the convulsions been general from the first, we should have concluded that the clotting began first in the sinus, and, if there was any clotting in the cerebral veins, that it was secondary to that in the sinus.

The cerebral sinuses may produce symptoms which, if far from pathognomonic, are often in a way suggestive of thrombosis of a particular sinus; the larger cerebral veins may also exhibit suggestive symptoms. But what of the veins of Galen when symptoms are not referable to the superior longitudinal, cavernous, and lateral sinuses? These veins are very likely to be thrombosed in purely marantic cases, also in erysipelas and puerperal septicæmia, and the patient may only exhibit such symptoms of illness as may be set down to his primal disease, with very possibly severe vertical headache and dimness of sight not due to any recognizable optic neuritis. Such a patient is very liable to suddenly exhibit the symptoms stated on the preceding page, which are similar to those which follow

the bursting of an abscess or cyst into the lateral ventricles; and post mortem the lateral ventricles will be found full of blood and recent clot found in the veins of Galen.

The **cavernous sinuses** are situated between the layers of the dura, one on each side of the body of the sphenoid bone. They extend from the sphenoidal fissure in front, where they are continuous with the ophthalmic veins, to the apex of the petrous bone behind, where they end in the petrosal sinuses. They are the site *par excellence* of infective thrombosis, caused by erysipelas, abscess, blebs, furuncles or anthrax on face or lips, and puerperal septicæmia. The next most important group of causes is infective inflammation of any of the accessory nasal cavities—mostly the sphenoidal—then caries of the body of the sphenoid and the ethmoid bones, and infective inflammation of the superior maxilla. The route of infection from the face and lips is usually by the facial vein, or, as in the case of the last-mentioned causes, by the ophthalmic veins, or by the perineural sheaths of these veins. Other routes are by the lymphatics and direct transference through the bone; and it may also become thrombosed by extension of the process from the sigmoids through the petrosals. The circular sinuses are very likely to be involved by the extension of the primal thrombus into them, and one or other must be if the cavernous sinus of the opposite side becomes affected. There is likely to be a localized meningitis over the affected sinus or sinuses.

Sir William Macewen says that no case is on record of this sinus being thrombosed from a marantic cause, and

this is probably a correct opinion, as the author has looked through the indexes of the medical weekly papers covering a period of thirty years, and found no case definitely stated to be marantic; however, No. 11 in our list had no assignable cause. Because no case is on record to refute the opinion that marantic cavernous sinus thrombosis does not occur, it does not necessarily follow that all examples of thrombosis of this sinus are purely secondary in origin.

Dr. Pigeon, of Hull, has recorded[1] a case, which I had an opportunity of seeing, in which, although there was no anæmia or other marantic cause apparent, there was probably a debased condition of the blood, due to an excess in it of calcium salts from a too exclusive milk diet, plus traumatism in the shape of an interference with the venous circulation, effected by a tight bandage having been applied round the head to support a fractured jaw.

Dr. Pigeon's case also provides us with an object-lesson in enabling us to contrast an aseptic cavernous sinus thrombosis with septic ones. In the former we have the objective symptoms of the condition well expressed, but no fever or brain symptoms, and after a protracted illness the patient practically recovers; in the latter, in addition to the objective symptoms, the patient is alarmingly ill, and the most probable ending is death within a week, and the most fortunate result we can look for is recovery after a long illness, with the loss of one or both eyes.

Symptoms.—The objective signs of thrombosis in

[1] *British Medical Journal*, December 12, 1908.

this sinus are associated chiefly with the eyeball of that side which becomes chemosed and pushed forward, the proptosis being caused by distension of the orbital vessels and the effusion present in the retro-ocular tissues; the cornea becomes hazy and dry, making ophthalmoscopic examination difficult or impossible, but optic neuritis is likely to be present, the eyelids and skin of the affected side of the face become œdematous, and in bad cases the free edges of the lower lids may be overlapped by the red and fleshy chemosis of the ocular conjunctiva.

Severe pain is apt to come on suddenly, and is situated at the vertex, in the forehead, or in or about the eyes; the pulse is quick, temperature high, and rigors may occur; there is often delirium, but the mind may remain clear. In cases Nos. 32 and 39 there was basal meningitis; in another case the superior surface of the brain was covered with pus; in No. 15 there was hemiplegia, and paralysis of one limb only may be present. The objective signs are seen at first only on one side, but the condition is very liable to become bilateral by extension of the thrombus of, or infection through, the circular sinuses. The patient's state is always most serious, and the majority of people die. If recovery takes place, it will be with the loss of one or both eyes, or the patient may, in rapidly fatal cases, die before the objective signs develop; and if the disease is correctly diagnosed in such cases, it will be from the severity of the general symptoms, coupled with the knowledge the doctor possesses that infective thrombosis of the cavernous sinus is liable to occur in certain diseases.

Lateral Sinus, including the Sigmoid Sinus.

Secondary thrombosis of these sinuses is more frequent than that of any other of the cerebral sinuses, and occurs most often during early adult life.

Ætiology.—The chief cause is the extension of chronic purulent discharge from the middle ear; less frequent causes are the extension of acute inflammation of the tonsils and posterior nares along the Eustachian tube into the middle ear; extension from other sinuses, usually the superior petrosal, less often the straight; and from the internal jugular vein, or from septic states of the mastoid process.

Symptoms.—There is no one sign or symptom which enables one to say that the condition of a patient is due to infective thrombosis of the lateral or sigmoid sinus, but several symptoms are very suggestive, particularly when they are in association. The most important are an irregular temperature, rigors, persistent diarrhœa, and profuse sweating. The objective symptoms are tenderness and œdema over the mastoid, tenderness, œdema, and pain on pressure being particularly marked over the site of the emissary vein of the mastoid (Greisinger's symptom), and a painful, swollen, and whipcord-like state of the internal jugular vein, felt on the inner side of the sterno-mastoid muscle. The last-named symptom may be absent, and it may be present when the blood in the lateral and sigmoid sinus is fluid. 'In the same neighbourhood there may be a general fusion of muscles and enlarged glands, or a localized abscess under the deep cervical fascia in the upper third of the posterior cervical triangle' (Macewen).

The general symptoms are early and severe headache, referred often to the side of the causal disease. Sir William Macewen strongly emphasizes as a symptom of this form of sinus thrombosis 'pain in the ear, described as darting through the brain from one ear to another.' Frequent vomiting, dry and coated tongue, offensive breath, absence of appetite, and very likely diarrhœa. The pulse is quick, weak, and thready; the temperature rises quickly to 103° or 104° F., and runs a peculiarly irregular course; rigors appear and recur frequently, often at the same hour on the following or second day, and therefore looked upon as of the ague type, and are always followed by profuse sweats; and if these symptoms are all well marked, and then vertigo and nystagmus appear, they may be regarded as indications that the petrosal sinuses are also occluded, or that one or more of the semicircular canals are injured or destroyed, and much blood mixed with the aural discharge points to erosion of the wall of the sigmoid sinus. Thrombosis of the sigmoid sinus is not necessarily a septic one when there is foul pus in the mastoid cells, and patients may recover without the sinus being opened: nor is the presence of an extradural abscess in immediate contact with the sinus absolutely conclusive that the contents of the sinus are septic; but if after mastoidectomy there is not a marked remission in the severity of symptoms, particularly in the fall of temperature, it is very likely there is infective thrombosis.

Macewen mentions that, in addition to the general symptoms, there are others which affect chiefly the lungs, intestines, or cerebral meninges, of which the

following, according to that author, are the chief signs:

A **pulmonary** type, producing cough, pain in the side, not stationary in one particular place, dyspnœa, prune-juice expectoration, which later becomes very offensive in odour.

An **abdominal** type, of which the symptoms are typhoidal in character, but the objective signs in the neck differentiate the disease from typhoid fever.

A **meningeal** type, marked by severe frontal headache, vomiting, seldom rigors, great excitement and hypersensitiveness, and severe girdle pains when leptomeningitis is spinal as well as cerebral.

CHAPTER III

INTRACRANIAL ABSCESS

INTRACRANIAL abscess will first, for simplicity's sake, be considered as if it were a disease which occurred on all occasions without association with other intracranial diseases, which is not by any means the rule, particularly when the abscess has resulted from middle-ear disease, in which case purulent leptomeningitis or infective sinus thrombosis, or both, may occur along with the abscess and mask its presence. Afterwards, as opportunity offers, its connection with the complicating diseases will be referred to.

If a person has for some time exhibited symptoms of intracranial tumour, and then has those symptoms increased in severity, and develops a febrile temperature of several degrees where previously there was no fever, with possibly a rigor, there is *prima facie* evidence that the tumour is breaking down into abscess.

Cerebral and cerebellar tumours may coexist, but it is not often that both are diagnosed in the period before operation, the symptoms of the one overriding those of the other. What usually takes place is that the one of which the symptoms are most obvious is first dealt

with, and, its special and characteristic symptoms disappearing, the patient remains practically unrelieved unless and until the other abscess is diagnosed and receives attention. Sometimes, unfortunately, the actual state of affairs becomes known only at the autopsy.

If we find that our patient has developed cerebral symptoms after otitis media, let us remember that pyæmia, meningitis, thrombo-phlebitis of the cerebral sinuses, usually the lateral sinus, and subdural abscess, are far more likely to be present than abscess of the brain itself, and that meningitis is the disease which usually ensues upon acute otitis media. Meningitis and abscess may either of them follow an attack of influenza, but we have valuable assistance in cyto-diagnosis that we did not possess a score, or even a dozen, years ago.

The facial appearance of a person suffering from intracranial abscess is one of deep distress, and conveys to the onlooker the idea of serious illness.

The first symptom of intracranial irritation following upon aural infection, whatever the resultant trouble, may be enlargement of the deep cervical glands, which escape notice unless especially looked for, as they lie under the deep cervical fascia.

Although some symptoms, such as the tâche cérébrale, retraction of the abdomen, and an increased leucocytosis, may point to meningitis only, there remains room for doubt when focal signs are indefinite, and it is remembered that the general condition in the one disease agrees with that in the other; also that Kernig's sign, if present, may strongly influence our minds against abscess, because it is chiefly a meningeal symptom,

and has also been met with in uncomplicated cases of abscess. When one is dubious on the question, it may be well to remember that constipation is common in meningitis, and diarrhœa is equally so in abscess and infective sinus thrombosis.

The points of difference between cerebral and cerebellar abscesses will be adverted to in another place, but it may here be stated that non-purulent encephalitis, septic meningitis, and mastoiditis, have all been diagnosed as abscess.

Surgical procedure may be futile, because, although abscess of the brain has been correctly diagnosed, mistake has been made with regard to its *locale*. The wrong hemisphere has been operated upon, often the wrong lobe, and, in case of encysted abscess, the containing wall may be so thick and elastic that the exploring needle or trocar glides along the side of it instead of piercing it. The pus may be too thick to pass through the needle, or the bore of the latter does not allow of the passage of the sloughs, which are frequently mixed with the pus, particularly in cerebral abscesses, or it may be a case of diffuse purulent encephalitis with little pus, or the drainage that is adopted may not empty some distant pocket; there may be a secondary abscess, quite distinct from the one that has been opened, or possibly the localized red softening has not yet entered upon the suppurative stage, the operation, indeed, being a little too premature.

Naturally the most striking or significant symptom occupies the observer's mind very much to the exclusion of others, and while in the majority of cases it is

correct to be thus influenced, it is at times a most unfortunate incident, for the significant symptom may not be caused by direct or immediate pressure upon the centre that gives the warning, but by a cause somewhat remote. *E.g.*, a person falls and injures his forehead. In a period which may run into weeks, months, or years, there is developed a twitching, weakness, or paralysis of arm or leg, or both of them, and in consequence the motor area known to contain 'the centres' for the extremities is pitched upon as likely to be the site of the lesion. The brain is opened, with negative results, and at the necropsy the abscess or tumour is found in the prefrontal lobe, the significant symptom being caused by transmitted pressure. Even if by good fortune the actual *locale* of an abscess is found after repeated explorations, it cannot be to the patient's welfare to pass through the ordeal.

Similarly, with regard to the cerebellum, symptoms highly suggestive of cerebellar abscess are developed, and first one and then the other side is operated upon without pus being found, but post-mortem examination reveals abscess in the optic thalamus or crus cerebri.

The moral of such mistakes seems to be that, however strongly one symptom may point to a given region of the brain as the site of lesion, subsidiary or lesser symptoms and facts should always receive due attention.

Elsewhere attention will be drawn to the fact that hemiparesis, hemiataxia, and hemianæsthesia, occurring singly or in association, are, when they occur on the *same* side as a facial paralysis, truly suggestive of a cerebellar origin; and it is mentioned here in order to

emphasize the statement that these conditions *must* be homolateral to the lesion, in contradistinction to the cases in which there is a cranial nerve paralysis upon one side and a hemiplegia, etc., upon the opposite side, when the lesion is sometimes in a site beyond operative interference.

Abscess may easily be, and often is, diagnosed when only great intracranial hypertension exists, and the cause of which may never be discovered; yet the operative procedure undertaken for the one is curative of the other, but not always. This statement holds good for the mistaken diagnosis of cysts and tumours as well as abscess.

There are no symptoms which can be looked upon as suggestive of the probable size of an abscess, as a small one may produce as serious an illness as a large one.

A mistake which operators are very prone to make is that when they trephine for supposed intracranial abscess and find the dura mater normal in appearance and the brain pulsating, they conclude, often wrongly, that the trouble is not in that particular region, and hold their hand from further progress. After-events have often proved that this has been a fatal mistake, and a more courageous policy would have been probably a happier one for the patient; and the rule would be a good one that if localizing signs are unequivocal, the condition of the dura should be ignored and the search for pus continued, as a dull and grey dura and non-pulsating brain are only indicative of pus at or near the surface.

An occasional source of failure is that when the surgeon opens the skull and finds a collection of pus

between the bone and dura mater, he concludes that he has found the *fons et origo* of the symptoms; and he may find in a very short time—which time may be too late—that there was also a deeper trouble. If in such cases there seems to be nothing in the appearance of the dura or in its bulging forward to denote intracerebral or intracerebellar abscess, the case should be closely watched, and if, after the effects of the anæsthetic have quite passed away, the previous more obvious symptoms, such as headache, paralysis, or aphasia, continue still in evidence, or the patient have a rigor, no time should be lost before the suspected lobe be explored.

Brain abscess due to the breaking down of a gummatous or tuberculous deposit stands forward in a light that is often denied to the otitic abscess, in that the general and focal symptoms stand out in all their significance, unqualified and unmodified by the symptoms produced by the complications common to the latter.

Melancholia has been found to be dependent upon intracranial abscess, usually the parietal lobe, without any of the usual symptoms produced by such abscess being present. A case is recorded by Mr. Arnold Jones of a woman who suffered from hallucinations and melancholic stupor; eventually a diffuse external otitis appeared, and the external auditory canal was incised. The ensuing suppuration being considered too copious for a localized necrosis, the radical mastoid operation was performed, and a sinus was exposed leading through the mastoid cells to the sigmoid groove, in which situation an extradural abscess was found and evacuated, and the melancholia and hallucinations disappeared.

All intracranial abscesses do not kill if the surgeon's skill is not brought into action; but cases of recovery without operation—by the combined effect of absorption by the small vessels which pierce the abscess capsule, and the pressure of the surrounding brain matter in the case of small abscesses, or finding an outlet either through a perforated tympanum or the eroded skull in the case of larger ones—are so rare that it would be in the last degree unwise to place any reliance on the probability of such a natural ending.

The fact of more pus issuing from the external meatus than can be accounted for by suppuration in the tympanum or mastoid cells may, as in the preceding case, afford the only clue to brain abscess.

The most severe general symptoms of brain disease may only be due to necrosed bone, and disappear when the dead bone has been removed, an illustration of which most of us have seen in necrosis of the mastoid bone after scarlet fever (in a case of this kind that was once under the author's notice the rapid and extreme emaciation was most obvious, and the quick putting on of flesh after dead bone was removed was equally noticeable); or it may be observed that after scarlet fever or measles the child continues to have an irregular temperature, does not take his food well, and loses rather than regains strength.

When the medical advisers have succeeded in making the fact quite apparent to themselves that intracranial abscess exists, and they can with some certainty define its site, it becomes their duty to press forward the question of operation, and the urgent necessity of

immediate action should be urged upon the patient or friends with all the tact and persuasive power at their command. The fact may be dilated upon that delay has a twofold danger: first, that imprisoned, and probably foul, pus is rapidly poisoning the system and reducing the patient's recuperative power, if operation be eventually performed, to the lowest minimum; second, that at any time the abscess may burst its bounds and destroy life by surrounding the healthy parts of the brain with its offensive and putrid contents. If the abscess is supposed to be cerebellar, it may be pointed out that there is very dangerous pressure being exercised upon vital portions of the nervous system, and a sudden cessation of breathing may occur at any moment; and if this untoward accident should happen during operation—and the longer operation is delayed, the more likely is such stoppage of breathing to occur —the operator will be seriously inconvenienced, and the patient's life probably thereby sacrificed.

All times during the pyogenic process which culminates in the formation of stinking pus in the brain are not equally opportune for surgical interference, as we will shortly explain.

In addition to the difficulty of obtaining consent for operation during the most favourable time, the medical advisers may be prevented from arriving at a definite opinion in many and diverse ways: some symptom they have always been accustomed to associate with brain abscess is wanting, or it is not expressed in a way that is sufficiently forcible to them; and one of the most fatal mistakes a man can make is not to operate

because there is no optic neuritis ascertainable by the ophthalmoscope; in regard to optic neuritis, it should always be borne in mind that, although neural inflammation has been found even after a few days' illness, as in a case of fulminating epidemic cerebro-spinal meningitis, it is a condition that may take weeks, or in case of tumours months, in which to make itself apparent.

We may here seek to emphasize the fact that if operation is carried out during the inflammatory stage, while the pupils are still quickly responsive to light, and the patient is irritable and stupid rather than sleepy or stupose, and the pulse is quick and temperature regularly febrile, the surgeon may not come upon abscess, but in a day or two he may find pus trickling from the trocar aperture.

The period *par excellence* when surgical interference will have the most favourable result is the early purulent stage, when intense general headache and high fever have remitted, and irritability and stupidity have been displaced by the stupose or sleepy state; when the senses have become dulled and cerebration is slow; when one pupil is more widely dilated than the other, but not very widely dilated, and the temperature is either just above or below normal, and the pulse is somewhat slow. With the closing of this stage the patient's chances of survival after operation rapidly retrogress. If operation has been delayed until the **paralytic** or **toxæmic** period has arrived, it will be entered upon with such feelings as the soldier may have when he undertakes a forlorn hope. **Both** pupils widely dilated or contracted,

deep coma or loss of power of speech and thought, the passing of urine and fæces involuntarily, intense optic neuritis, and Cheyne-Stokes or stertorous breathing, constitute a syndrome of symptoms which are in the highest degree suggestive of the vital powers being at their lowest, and are forewarners of an unhappy result if the offending matter be not removed; but even now brilliant work has been done, and persons have been rescued when at the very edge of the grave. At this juncture the examination of the blood and cerebro-spinal fluid will be very helpful as throwing light upon the chances of recovery. If there is a high leucocytosis of the blood, the condition is favourable even if the symptoms are severe; but if there is a low leucocytosis of the cerebro-spinal fluid and many polymorphonuclears, with much albumin, the case is unfavourable for operation.

The last words in the preceding paragraph lead on to the all-important question, Is the abscess operable? If the patient is in hospital, and the fact is patent or very possible that the lesion is in or near vital centres, or cannot be reached, the futility of operation has to be explained to the patient or friends; and if the patient lies at his own home, it would be insensate to bring an operating surgeon, perhaps a long distance, and therefore at great expense, only for him to have to say that operative interference is impossible. Thus great responsibility rests upon the *medical* advisers to eliminate, if they can, the idea of inoperability, and the pages of this work devoted to a short summary of the signs and symptoms of lesions situated in inoperable areas will not be the least in importance.

The symptoms of cerebral and cerebellar abscesses are to a certain extent very similar—that is, the general symptoms of the early stages are similar; and in a less degree they both coincide with cerebral meningitis—be it simple or tubercular—typhoid fever, uræmia, and sometimes lead-poisoning.

On account of this general similarity, the ætiology, pathology, and symptoms (in stages) will be considered under the heading of cerebral abscess, which includes the great majority of intracranial abscesses, and the points in which cerebral and cerebellar abscesses differ will be pointed out as each symptom in the two varieties is under review.

Brain abscesses traceable to ear troubles can with much precision be located in the cerebellum, or to a collection of pus lying beside it in the occipital fossa and in the temporo-sphenoidal lobe of the cerebrum, or an extradural collection of pus lying on the posterior half of that lobe, because these parts of the brain lie nearest the temporal bone in which aural suppuration occurs; and it is said that with such a cause the cerebrum is the site rather more than twice as often as the cerebellum.

Cerebral Abscess.

Definition.—Abscess of the brain may be defined as a purulent encephalitis, sometimes diffuse, with little pus, but more often as a localized and delimited abscess altogether within the brain or on its surface, with the outer wall formed by the inflamed and thickened meninges.

Age and Sex.—Cerebral abscesses occur most often during youth and early manhood—*i.e.*, the period between ten and thirty years of age—the second decade of life accounting for the great majority of them. Infancy and childhood include a good many cases, but none of very tender years are in the appended list of fifty-nine cases, the youngest in which were a boy and a girl, each being nine years old, both recovering. At the other end of the line we find matters less hopeful, for out of six cases ranging from forty-three to fifty years, three died. The disease is far more frequent in males than females:

	Females.	*Males.*	
Cerebrum - -	13	36	or 1 to 2·76.
Cerebellum - -	4	26	or 1 to 6·5.

This preponderance may be partly accounted for by the fact that males are far more likely than females to be the subjects of blows, kicks, and other forms of violence; but that traumatism is only in part the cause of this great disparity is seen in the large number of cases in males which arise in connection with ear troubles after the zymotic diseases, to which boys are no more subject than girls.

Ætiology.—Abscess of the brain is always considered as secondary to some pre-existing disease, chiefly pyæmia and inflammation of the middle ear, and injury; but the possible causes are many, and they admit of a classification under the two chief heads of local and distant.

1. *Local causes* include cranial suppurations and injuries to the head.

2. *Distant causes* include distant suppurations and infective foci carried to the brain from certain systemic diseases.

1. Local Causes.—Cranial suppurations may be due to caries in the bones of the nose and its accessory cavities, but these only cause a fractional portion of brain abscesses, disease of the middle ear and pus in the mastoid cells accounting for from one-third to one-half of intracranial abscesses, including almost *all* cases of cerebellar abscesses; therefore the consideration of causes other than aural come under cerebral abscess only.

Although an otorrhœa may be of months' or even of years' duration, with consequent perforation, and perhaps complete destruction, of the tympanic membrane, cerebral abscess has been known to follow an otorrhœa of only two weeks' standing, and to occur when there has been no perforation of the drum; and while the discharge usually ceases before the development of brain symptoms, it is not a necessity that it should be so. On the other hand, a recent otitis is more likely to produce meningitis than brain abscess, because in a short time the meninges do not become fused together, and infective material easily enters the subarachnoid space. Acute otitis media may follow, or be intensified by, an acute catarrh of the upper air-passages or a blow upon the side of the head; be a sequel of scarlet fever or measles; be an extension from an acute tonsillitis or the local inflammation started by an aural polypus; and in children, after the exanthems, the chief starting-point is in adenoids.

It may be difficult to determine the primal cause of

abscess, as there may be a local cause, as an otorrhœa, and a distant cause, as bronchiectasis, just as we have mentioned elsewhere there may be two causes working together, either of which may be causative of acute meningitis.

Scarlet fever and measles are, by their production of otorrhœa, the most frequent causes of diffuse purulent meningitis, lateral sinus thrombosis, and intracranial abscess, whether extradural or within the brain; and of these two diseases, scarlet fever takes the premier place, one-half to two-thirds of intractable otorrhœas owing their origin to it.

'Zymotic' otitis differs from the 'ordinary' or 'catarrhal,' both with regard to the causal micro-organism and its route to the middle ear and the parts of the ear upon which it exerts its destructive energy. The micro-organism of the zymotic is the *Streptococcus pyogenes*, which enters from the throat and reaches the tympanum by the Eustachian tube, producing an inflammation of the whole of the auditory tract, electing the mastoid cells and antrum upon which to work its greatest injury, and there developing a purulent mastoiditis. Sometimes it gives rise to an osteitis, while the catarrhal originates from the staphylococcus or pneumococcus, produces a catarrh, which does not extend beyond the tympanic cavity, and is not associated with osteitis.

The route of intercommunication between ear and brain is through the **tegmen tympani** or **antri**, and is explained by Macewen by the fact that the temporo-sphenoidal lobe lies upon the roof of the tympanum—a

thin plate of bone, and therefore the more easily eroded —which is pierced by veins emptying themselves into the superior petrosal sinus, and in case of mastoid suppuration into the lateral sinus, by which and the perivascular lymph channels infection is carried to the brain. It has also been suggested that in the young the route may be by the band of connective tissue which binds together the squamous and petrous portions of the temporal bone, but that the routes by the **fenestræ ovale** and **rotundum**, which one would imagine to be the most likely, are not often taken.

Other forms of suppuration which may cause cerebral abscess, but seldom so, are those of the nose and its accessory cavities.

A more frequent cause is caries of the temporal bone. Caries of other cranial bones are possible, but rare, causes.

Another infrequent cause is the breaking down of a tuberculous or gummatous growth already existing in the brain, for an example of which readers are referred to a case reported in the *British Medical Journal* (May 2, 1903) by Dr. Dutt and the late Dr. Lowson of Hull.

Violence in one shape or other accounts for a large number of cerebral abscesses—stated to be from one-sixth to one-fourth of the whole, and second only to ear disease.

The injury may have been slight, and on that account forgotten, and, having produced no breach of the surface, has left no cicatrix or other external sign to assist localization of any lesion; or it may have been of a severe character, causing fracture, perhaps unrecognized, the ultimate result being necrosis of the inner table of the

skull and extradural abscess; or the brain below the site of injury may have been bruised, the bone itself escaping. When the injury is a fall or blow, or a blow with a heavy and blunt instrument, the abscess is superficial—meningo-cortical—the infection being carried from the injured bone by a diploëtic vein. The injury may be a punctured wound, the weapon or instrument penetrating the brain itself carrying the infection, such as a woman's hat-pin; or a knife; scissors, open or closed; a hook, such as butchers use to hang meat upon; or the heavy and pointed hook in use by dock labourers; or be made by the bullet from revolver or rifle; or be a punctured and lacerated wound, as in the case of spicula from a fracture; or perhaps, and worst of all, a poisoned and punctured wound made by falling upon a projecting rusty nail. As none of these puncturing agents are likely to have been sterilized for the purpose, and are sometimes not ordinarily clean, they are likely to introduce micro-organisms more or less deadly, and it is therefore surprising that this class of injury is not always followed by abscess; but such is by no means the rule.

The most remarkable case of this kind of immunity from serious after-consequences the author has heard of was that of a boy in Lincolnshire, who was employed by a farmer to scare birds from corn. While the youngster was pressing home a charge of powder the gun went off, the ramrod passing obliquely through the brain, entering the skull at the forehead and leaving it by the parietal bone.[1]

[1] Dr. Ferrier quotes a much worse case in 'Localization of Cerebral Disease.'

When cerebral symptoms follow on after injury to the skull, the resulting diseases admit of a classification like the following, according to the time the symptoms appear after the accident:

(*a*) When symptoms come on at once or in a few hours, there is subdural hæmorrhage or laceration of the brain. It has been denied that there can be any laceration of the brain without fracture of the skull, but two cases I saw some years ago prove the contrary.

A boy, six years old, was riding a polo-pony out for exercise, and cannoned against a horse harnessed to a milk-cart; he lost his seat, and fell with some violence with his head upon one of the shafts. I saw him in an hour, and found him unconscious, but his friends said that he had only become so within the last few minutes. He shortly began to breathe stertorously, had unilateral sweating, and died within six hours of the accident. On post-mortem examination no fracture of the vault or base of the skull was found, but there was much laceration of the brain and hæmorrhage.

A man of middle age was descending the ladder of an omnibus, which swerved and caused him to fall, but the violence of his fall was lessened by his head coming on to the steps before he reached the ground. He rose, and with assistance walked a short distance to his home, but felt dazed. He had a restless night, and in the morning began to vomit badly. I saw him about 11 a.m.; he was then quite conscious, could express himself coherently, had full use of his limbs, and there was no hæmorrhage from ears or nose. During the day he had much headache, became drowsy, the drowsiness deepen-

ing into coma during the night; next morning he was breathing stertorously and was hemiplegic, and died thirty-one hours after the accident. On post-mortem examination there was found no fracture of the skull, but much laceration of the brain and hæmorrhage. My opinion was that the fall caused only a slight laceration, but the vomiting and retching caused further hæmorrhage and consequent ploughing up of the brain.

(*b*) When symptoms appear a few days or weeks afterwards, you may expect encephalitis or abscess.

(*c*) When symptoms develop months or years after an accident, the lesion will be traumatic cyst, adhesions, with or without depressed bone, or malignant tumour. These rules can only be accepted as portraying the usual course of events, as it will be seen that the following cases seriously invalidate them:

On July 2 a case was reported in the *Lancet* in which serious brain symptoms did not arise until many weeks after an accident. At the end of seven weeks the skull was opened, and effused blood was found to be the only cause of the symptoms.

A man fell from his bicycle and received a scalp wound, which healed; but he continued vaguely ill and depressed, and not until ten weeks after the accident did pressure symptoms arise. He was trephined, and the dura mater was incised; several ounces of blood escaped, and some clot was found adherent to the pia-arachnoid. Improvement was immediate.[1]

When any length of time after an accident symptoms indicate that a large area is affected, the trouble is

[1] *Lancet*, September 19, 1908.

either a large clot of blood, multiple abscesses, multiple cysts or tumours, or extensive adhesions.

2. Distant Causes.—These include infection from general systemic and organic diseases and distant suppurations. Diseased conditions in distant parts of the body produce septic material, which obtains entrance into the blood-stream, and is eventually deposited in the brain.

The general systemic and organic diseases which have caused abscess of the brain are pyæmia, influenza, enteric fever, smallpox, valvular diseases of the heart, hepatic inflammations, and badly resolving pneumonias; but all authorities are agreed that tuberculous disease of the lungs is little likely to cause abscess. Shorstein says that cerebral abscess occurred only twice in ten thousand cases of lung disease in the London Hospital.

'It has been denied by some writers that influenza can be the cause of cerebral abscess, but there is overwhelming proof of such capability, and such proof lies in the fact that the specific organism of influenza has been found in the pus of cerebral abscess' (Grunbaum).[1]

Out of this list of diseases pyæmia furnishes the most examples of cerebral abscess, and they are mostly multiple. Of them, Sir William Gowers says: 'Pyæmia never, and distant suppurations rarely, cause cerebral abscess in persons under twenty years of age.'

The distant suppurations are simple fœtid bronchitis, bronchiectasis, abscess of the lungs, caries of ribs, empyema, gangrene of lungs, fœtid pericarditis, and infective compound fractures.

[1] 'Cerebral Abscess,' in Nothnagel's 'System of Medicine.'

Abscess due to influenza, pyæmia, diseases of the bronchial tubes, lungs or pleuræ, caries of the bones of the skull (due to violence, cancer or syphilis), may occur in any part of the brain, as also may those due to such rare causes as actinomycosis and *Oïdium albicans*.

The analysis of the causes in the fifty-nine cases is:

Otorrhœa	34
Caries of temporal bone	1
Pulmonary diseases, in three of which the pneumococcus was found	5
Syphilis	1
Actinomycosis	1
Nasal catarrh	1
Influenza	2
Ill-health	1
No cause known	1
Violence:	
Fall from ladder	1
Fall upon back of head upon ice	1
Perforating wound of skull	1
Boiler explosion	1
Blow on forehead with bat	1
Severe blows on head	7
Total	59

A further examination shows the hemispheres to be pretty equally divided as to the site of abscess, the preponderance of males over females, and the associated disease, if any.

No.	Cause.	Site.	Age in Years. M.	Age in Years. F.	Associated Disease.
1	Otitis media	L. temp.-sphen. lobe	—	—	Mastoiditis.
2	„	R. temp.-sphen. lobe	—	—	„
3	„	R. „ „	30	—	Caries of temporal bone; lepto-meningitis.
4	„	L. „ „	—	—	Mastoiditis.

No.	Cause.	Site.	Age in Years. M.	Age in Years. F.	Associated Disease.
5	Otitis media	L. temp.-sphen. lobe	12	—	—
6	„	L. „ „	—	10	Thrombosis of lateral sinus.
7	„	R. temp.-sphen. lobe	19	—	—
8	„	L. „ „	50	—	—
9	„	R. „ „	19	—	Purulent pachy-meningitis.
10	„	L. temp.-sphen. lobe	—	9	—
11	„	R. temp.-sphen. lobe and R. frontal lobe	—	16	—
12	„	L. temp.-sphen. lobe	48	—	—
13	„	L. temp.-sphen. lobe, two distinct abscesses	—	—	—
14	„	R. temp.-sphen. lobe and occipital lobe	—	—	Encephalitis of left hemisphere.
15	„	L. temp.-sphen. lobe	30	—	Epidural abscess.
16	Otitis media, double	R. frontal lobe	28	—	—
17	Otitis media, double	R. temp.-sphen. lobe	—	21	—
18	Otitis media	L. „ „	—	14	—
19	„	L. „ „	22	—	—
20	„	L. „ „	17	—	—
21	Otitis media, double, after measles	L. „ „	17	—	Mastoiditis.
22	Otitis media	R. „ „	—	—	—
23	„	L. „ „	31	—	Mastoiditis and lateral sinus thrombosis.
24	Otitis media, double	R. temp.-sphen. lobe	47	—	—
25	Otitis media, double	R. „ „	—	20	Mastoiditis.
26	Otitis media, double	L. „ „	14	—	„

No.	Cause.	Site.	Age in Years. M.	Age in Years. F.	Associated Disease.
27	Otitis media	L. temp.-sphen. lobe	16	—	—
28	„	L. „ „	26	—	—
29	„	L. „ „	47	—	—
30	Otitis media, after scarlet fever	R. „ „	33	—	—
31	Otitis media	R. „ „	—	19	—
32	„	L. „ „	33	—	—
33	„	L. „ „	14	—	—
34	„	L. „ „	14	—	—
35	Otitis media and traumatism	L. „ „	26	—	—
36	Traumatism	Temp.-sphen. lobe	—	—	Mastoiditis
37	„	L. temp.-sphen. lobe	15	—	—
38	„	R. „ „	18	—	—
39	„	L. „ „	14	—	—
40	„	L. prefrontal lobe	43	—	—
41	„	R. temp.-sphen. lobe	15	—	—
42	„	Extradural, between dura mater and temp. bone	38	—	Mastoiditis.
43	„	Frontal lobe	—	—	—
44	„	R. frontal lobe	11	—	—
45	„	R. temp.-sphen. lobe and frontal lobe	—	—	—
46	„	R. frontal lobe	11	—	—
47	Disease of lung	Greater portion of right hemisphere and left temporo-sphenoidal lobe	18	—	—
48	Abscess of lung	L. temp.-sphen. lobe	46	—	—
49	Tubercle	L. parietal lobe	—	—	—
50	„	L. cornua of lateral ventricles and radiations	—	—	—
51	Influenza	R. frontal lobe	—	—	—
52	„	L. „	—	24	Purulent basal meningitis.
53	Ill-health	L. „	—	17	—
54	Empyema	L. post-frontal lobe	—	—	—
55	Actinomycosis	R. frontal and parietal lobes	—	—	—

No.	Cause.	Site.	Age in Years.		Associated Disease.
			M.	F.	
56	Caries of temporal bone	R. temp.-sphen. lobe	47	—	—
57	Nasal catarrh	L. parietal lobe and centrum ovale	—	16	—
58	No cause known	R. frontal lobe	—	—	—
59	Syphilis -	L. „	—	—	—

Next to trauma and middle-ear disease, pulmonary disease (non-tuberculous) produces the largest number of cases of cerebral abscess, and these are usually in the left hemisphere, and more often in men than in women; and of pulmonary diseases, bronchiectasis—a relatively rare disease—takes first place, and empyema the next, whether it is left unrelieved, the pleura be drained by incision between the ribs or by section of a portion of ribs; gangrene of the lungs is a rare cause, so is acute pneumonia.

Oïdium Albicans.—Sir William Gowers mentions two cases of cerebral abscess caused by thrush in the mouth.

In one of the cases in our list no cause could be assigned. Most writers on the question of unassigned causes of cerebral abscess suggest that the cause may have been a blow or a fall, which was probably slight and did not cause much pain, and was soon forgotten; but whether cerebral abscess can actually arise without a cause—be, in fact, idiopathic—is a point that is not yet settled.

The *locale* of intracranial abscesses with reference to their causes may be summed up as follows:

The **temporo-sphenoidal** lobe, and chiefly its white

matter, is *par excellence* the site of abscess due to suppuration in the middle ear, and after it the **cerebellum**, but very seldom the middle lobe of the latter, and any form of violence which acts either directly or intensifies existing ear disease. The **frontal** lobe is much less often the site of an abscess due to otitis media than the temporo-sphenoidal region. Other causes are falls, or wounds on the forehead, possibly by *contre-coup* from falls on the back of the head, and not infrequently by septic infection from the nose and its accessory cavities.

In the **occipital** lobe we find that the abscess is due mostly to pyæmia, then to falls upon the back of the head, less often by *contre-coup* from blows on forehead, and very much less to otitis media than either the temporo-sphenoidal or frontal lobes.

Pathology.—Before abscess there must be softening. There are two varieties of this condition, the 'necrotic' and the 'inflammatory.' The former is known as the 'yellow,' 'white,' or 'green' softening, and is the kind which precedes dementia, general paralysis of the insane, etc. It most often occurs in the temporal and parietal lobes, corpus striatum, and optic thalamus, because the arteries there are terminal; and, there being no collateral circulation, when they become blocked by embolus or thrombus, the parts beyond, after undergoing molecular change, perish. The softening which precedes abscess is the 'red' or inflammatory form, otherwise localized encephalitis. It is started by infective foci introduced directly by the agent causing the injury—in case of otitis media by such foci being carried from the

middle ear by the small veins, which themselves may be in a state of septic inflammation, and by the perivascular lymph channels; or there is a septic meningitis, which infects the cortex of the brain by direct contact. The redness results from the distension of the capillaries and the extravasation of red corpuscles into the perivascular spaces. To the red softening soon succeeds a stage of initial suppuration, in which the affected part becomes swollen on account of the integral parts of the brain substance—nerve fibres, ganglion cells, and neuroglia cells—being separated by the exudation from the distended vessels of serum and leucocytes, which, with some pus cells formed from the cells of the neuroglia, constitute the pus. The colour is now yellow, and perhaps may assume a greenish tint, and the collection has at times a peculiarly offensive smell.

Abscesses may be of any size from a pea to an orange, and in rare cases—one occurs in our list—may involve a large portion of a hemisphere. Hæmorrhage may occur into an abscess, as it is known to do into the substance of a tumour, the symptoms then simulating those of apoplexy.

In case of pyæmia and distant suppurations, cerebral abscesses are usually multiple; when due to violence or ear disease, they are mostly single.

Some acute abscesses have no capsule, because the microbic poison is highly virulent, and the abscess runs its course without having the time necessary for the development of a capsule; but the surrounding brain matter in these non-capsulated abscesses is soft and œdematous, and the pia-arachnoid which covers it may

be reddened or opaque. If the abscess is chronic and grows slowly it may or may not have a capsule, but the absence of a capsule does not prove that the abscess is recent. When there is a capsule it is formed of connective-tissue elements—*i.e.*, granular cells and leucocytes—which have become entangled in the meshes of a fibrinous matrix, added to which there is pus and blood. It is thin at first, and becomes thick, tough, and elastic as time passes. When it has attained some thickness, small bloodvessels are seen to pass into it from the healthy brain; its inner surface is smooth, and the outer surface, which is contiguous to the brain, is irregular and rough. If there is a capsule, the contained pus, being shut off from the rest of the brain, is prevented from exerting any toxic injury upon the surrounding brain, particularly when, as sometimes happens, there is a firm layer of connective tissue outside the capsule. Improvement may begin and proceed until the focal and general symptoms disappear or become much less in evidence, or the symptoms by their chronicity simulate those of a quickly-growing tumour. But the day of reckoning is for the most part only deferred; the abscess may at any time rupture into the lateral ventricles or upon the surface of the brain, or injury to the head or further infection from the renewal of the aural discharge causes it to reassert itself in all, and very likely more than all, its former vigour. If neither of these eventualities occur, then, in the case of small abscesses, absorption of the more solid contents may take place, leaving a cyst or only a cicatrix behind, or the liquid contents may be absorbed, leaving a thick residue behind, and the capsule

may undergo calcification. A cerebral or cerebellar abscess usually runs its course in from ten days to five weeks.

Symptoms.—The pyogenic process ending in abscess of the brain, particularly when due to ear troubles, shows itself to the observer in three stages, often distinct, sometimes less so. They are:

First, the **inflammatory** or **febrile** stage, when the patch affected is in the state of red softening, and the symptoms are those of irritation, which lasts as a rule a few days, but at times is prolonged into a week, and usually follows the sudden cessation of aural discharge. The earliest symptoms may be influenzal in character: the patient has a slightly raised temperature and quick pulse; he complains of pains in his limbs and great weakness; his face is flushed; he has a furred or dirty tongue and may vomit; and after a day or two of this he begins to complain of a burning pain in the ear and paroxysmal or severe continuous headache, localized to the side of the head in temporal lobe abscess and to the occiput in cerebellar abscess, or the headache may precede all other symptoms. He becomes extremely irritable when spoken to; his hearing is bad, but he is not deaf, and he understands questions; he is inclined to drowsiness, but does not sleep; if he sleeps he is easily awakened, and there is stupidity, but not stupor. He is likely to have a rigor, probably bespeaking the breaking down of the red softening into pus, which will perhaps be severe, or, if frequently repeated, is suggestive of infective sinus thrombosis; the vomiting, which does not depend on the taking of food, continues, and

there may be no nausea. There is paresis rather than paralysis, and there may be aphasia, but it is unlikely at this stage; there is as yet no optic neuritis, and the pupils react to light and accommodation.

All these indications of poorliness may, in occasional cases, disappear; the disease, instead of progressing, becomes stationary, and a latent period of many months may elapse, when the walls of the abscess may give way, except in the very rare cases already spoken of, and death occurs with striking and almost dramatic suddenness.

The *second*, **manifest** or **early purulent**, stage is one of pressure, in which the symptoms characteristic of the first stage, such as headache, pyrexia and irritability, have remitted. The headache may be worse, but it is more likely to have passed away on account of the sensibility having become deadened; in any case, there is severe pain localized over the mastoid or squamous portion of the temporal bone, which is evidenced by a wince or other indication of pain when pressure is exercised upon it. Vomiting may now only be present on movement, but there is often nausea, and more often in cerebral than cerebellar abscess; and in the latter vomiting may still continue and vertiginous attacks are common, the tongue is thickly coated with fur, and there is no desire for food; the breath is extremely offensive, and the odour from it may be like that coming from the ear; and the bowels are constipated. The pulse is often much below normal in the number of beats, and may be weak and intermittent; the temperature has dropped to normal or below it, and if it is high

it probably means an associated leptomeningitis. The respiration is slow, markedly so in cerebellar abscess; paralysis is frequently present, and we have also present the usual evidences of an exhausting disease in the way of great and rapid loss of flesh, and possibly muscular tremors, and myoidema may be elicited. The face no longer shows intelligence and brightness; instead, we notice vacuous, expressionless, and meaningless features, and the patient's increasing weakness has compelled him to take to his bed, if he has not done so at a much earlier period.

Cerebration is slow, and he answers questions only after an interval; he may be unconscious, and perhaps moans as he lies with his hands on his head; drowsiness is passing into somnolence, and optic neuritis has developed and advances rapidly, and is perhaps more apparent on the diseased side. In cerebellar abscess choked disc is more often seen than optic neuritis. The reflexes will be exaggerated and the pupils will be sluggish or altogether inactive, and suddenly we may be warned of the oncoming of the third stage by a rigor, followed by profuse perspirations, several rigors having the same meaning as we mentioned above.

The *third*, the **terminal, toxæmic,** or **paralytic,** stage is one of pressure plus toxæmia, in which the pus has become green in colour and highly offensive, on account of the presence of those micro-organisms which do not require air or free oxygen for the support of life (anaërobic), and its effects upon the system are more obvious almost from hour to hour. The most characteristic symptoms of abscess are those of this stage, and

consequently it is often not diagnosed until these appear. The power of speech and thought may alike be gone, and there may be coma. Constipation has given way to diarrhœa, and urine and fæces are passed involuntarily. Optic neuritis may be intense; the breathing is very slow, stertorous, or of the Cheyne-Stokes character; the pulsations at the wrist may be as few as thirty per minute, and the temperature as low as 95° F.; the pupils are quite insensitive to light, and are either contracted or dilated, and unequally so, and deepening coma or general convulsions betoken an early fatal issue. In unrelieved cases death comes by deepening coma or rupture of the abscess; the latter event is very possible in abscesses which may have given no sign for a period or in which the symptoms may not have been at all urgent. If the abscess bursts on the surface of the brain, purulent meningitis is quickly developed from the meninges becoming infected through their contiguity with the peccant pus, and the patient begins to vomit, is restless and uneasy, has a flushed and anxious face, quick pulse, high temperature, and hurried breathing. When rupture takes place into the ventricles, the change in the patient's condition is startling and characteristic, and the ensuing symptoms may simulate those of ventricular hæmorrhage, and general convulsions, with immediate loss of consciousness, may occur; but usually it is noted that the pupils become suddenly widely dilated or contracted, the face becomes livid, and there is twitching of muscles all over the body; the pulse is quickened, the temperature rises from subnormal to hyperpyretic, the breathing

becomes hurried or stertorous, death occurring within two hours. If there is only an oozing of septic matter into the ventricles, the symptoms are not so urgent, but early death is just as certain, although life may be prolonged twelve or twenty-four hours.

A cyst may also rupture into the lateral ventricles, and cause a similar set of symptoms, as in the following case: Cerebral hydatid was diagnosed. The skull was trephined and the cyst drained. The patient did well for three days; the temperature then suddenly rose, and the patient died with marked symptoms of cerebral irritation. After death, the cavity in which the cyst had been was found communicating with the lateral ventricle.

The exceptions to cerebral abscess advertising its presence in more or less well-defined stages are the cases of latency to which reference has been made, and those cases which simulate an acute specific fever or acute delirious mania, in which symptoms develop with great suddenness, as when a person goes to bed ill and rises in the morning with severe headache, vomiting, and a high temperature, which is often found to be a chronic abscess with a firm containing wall of slightly organized material, which, harmless for months, has suddenly become dangerous from some form of traumatism or fresh aural trouble. In both these forms slight symptoms may have been overlooked, headaches being ascribed to other causes. The absence of symptoms until late in the disease may be explained by the lesion being at a distance from the motor and sensory centres.

The long list of focal symptoms which usually accom-

pany the growth of a tumour is curtailed often in the case of abscess, probably because toxæmia sets in before intracranial pressure has been present long enough to produce the various clinical phenomena which are common to tumours. As regards the general condition, we know that if a person suffers from cerebral abscess, malaise, dulness, incoherent speech, or great irritability may be the first points to attract attention, and the comatose state may to some extent be forecast by indistinct speech and inability to swallow solids, the dreamy or drowsy state, or transitory loss of consciousness.

Slow cerebration might well be printed in the largest type, for it occupies a foremost place in the symptomatology of brain abscess; it points to the senses having felt the deleterious influence of great intracranial pressure and some absorption of pus, and of the pus having become offensive, and consequently more poisonous—a change of things which may or may not be indicated by a rigor, but is always suggestive, not only of the necessity for immediate operation, but that the most favourable time for operation is quickly passing.

Retraction of the abdomen is an occasional symptom. It was markedly obvious in case No. 30, in which there was no meningitis, and both it and the tâche cérébrale may be taken as implying an advanced third stage, as in case No. 57, in which abscesses occurred in the left parietal lobe and centrum ovale; and incidentally it may be mentioned that a general twitching of the muscular system, especially of the muscles of the face, is a symptom pointing to the latter *locale* as the site of a lesion.

Insomnia is very characteristic; the patient may be only irritable, dull and stupid by day, but delirious and sleepless at night, although there may be no increase of temperature as a possible cause.

The patient may lie upon his back, with his knees drawn up, or he may on rare occasions assume the position which will later be mentioned as often being noticed in cases of cerebellar disease—of lying curled up on one side, and with his head supported between his hands.

In an acute abscess of some days' duration the lips may be livid, the tongue red and dry, and **sordes** be deposited on the teeth; but a cyanotic appearance of the face is an indication that the condition brooks no further delay.

While there is no symptom that can be definitely relied upon as fixing the size of the abscess, a wrinkled skin, deeply shrunken eyeballs, and a rapid emaciation, are suggestive that it is a large one.

HEADACHE.—Headache is often the first, as it is certainly the most pressing, initial symptom presented by anyone suffering from cerebral abscess, and it is present in most cases. The pain may be general and not severe, intense and general if the condition is a diffuse purulent encephalitis, intense and localized if the abscess is delimited, and it may be continuous or occur only in paroxysms; but however it is exhibited, it is likely to be mitigated when pressure symptoms become apparent. If there is tenderness on pressure or percussion, that fact is by itself a very valuable localizing sign, and is suggestive of the abscess not

being deep. If the pain is in the right temple, the abscess may be presumed to be located in the subjacent brain; but for the headache to be on one side of the brain and an abscess to be present on the other side would be in the last degree unlikely. Our list, however, includes one case of that description, and it is by no means uncommon to have severe pain in the forehead when the abscess is in the temporo-sphenoidal lobe.

When headache is frontal it is often of the intense order, and when it is experienced equally in the frontal, parietal, and occipital areas, the abscess may be supposed to be deeply seated.

As the volume of purulent matter increases, one would naturally suppose that the headache would become worse, but in practice this is not found to be the case—not only because rapid increase of cerebral hypertension has dulled sensation, but because there is another factor making itself felt—that is, the development of toxæmia—as the pus retrogrades and becomes absorbed.

Pain in the back of the head and radiating down the neck, with retraction of the head and rigidity of the cervical muscles, are not altogether unknown in uncomplicated cerebral abscess, but they are very rare as compared with cerebellar abscess. In case No. 29 there was retraction of the head, and events proved that there was neither meningitis nor cerebellar abscess; but it is probable that where pain at the back of the head and retraction are associated with cerebral abscess, some other intracranial disease is also present, and the most likely are purulent meningitis and cerebellar abscess, or

both of them. These associated symptoms were present in case No. 9, in which there was purulent pachymeningitis, and when this was relieved by trephining, etc., the stiffness and retraction disappeared; and although abscess was found in the inferior part of the parietal lobe a little later, a return of the stiffness and retraction did not take place.

If pain is elicited by percussion over one particular part of the head, there is good reason to believe that there is disease under that particular place, but the disease is not necessarily cerebral abscess.

Vomiting.—Vomiting, sometimes associated with great nausea, is common in cerebral abscess, but not characteristic of it, and it is more likely to be an occasional symptom, perhaps only being noticed two or three times during the course of an illness. The tongue is often thickly furred; at other times it is clean in the early days of the disease, vomiting being then absent, and becoming red, dry and glazed at a later date. With either condition the odour of the breath is a peculiarly sickly one, and is characteristic of the disease; an extremely offensive breath may denote cerebral abscess plus sinus thrombosis.

If there has been obstinate vomiting in connection with an abscess between the bone and dura, and evacuation of the pus may not cure it, it may be found later that there is also abscess in the cerebral substance.

A confined state of the bowels is looked upon by many practitioners as a necessary concomitant of cerebral abscess, but it is by no means always so. The bowels may be regular throughout the illness, or if the

abscess goes on unrelieved, looseness is present some time before death.

Unless vomiting, with a thickly furred or dry, red and glazed tongue, be present, when food will necessarily be distasteful, the appetite may continue very good, in spite of apathy and drowsiness.

VERTIGO.—Vertigo may also be a marked symptom, and while it may occur in abscess of the temporo-sphenoidal lobe, its presence is more likely to point to the internal capsule.

PULSE.—The pulse is quick in the inflammatory stage, but at the period when cerebral abscess is likely to be recognized as such its characters are slowness, weakness, and irregularity. In the number of beats it is, after the first stage, normal or only a little slower than normal at first, but slows as stupor deepens, until there may be only forty beats per minute, or even only thirty. Eighty beats per minute constitute a quick pulse for cerebral abscess, except in the very last stage, when there is a marked tendency for it to become very rapid. That the pressure on the surrounding brain substance is the predominant factor that causes an abnormally slow pulse, and not the toxæmia the abscess has engendered, is demonstrated by the fact that when a cerebral abscess is opened the pulse quickens as the abscess empties itself, although it may again slow in an hour or two. This slowness of the pulse is usual, but not by any means absolutely general; in one of our fifty-nine cases the pulse was 106 and the temperature 100·8° F. during the early purulent stage.

TEMPERATURE.—A high temperature is not a marked

feature, except possibly in the initial stage. During the second and third stages of the disease the temperature is normal or subnormal, and there may be no pyrexia at all when the trouble has so far advanced that the patient is in a perilous state. If there is an increase it is slight, not more, perhaps, than one or two degrees, except just after a rigor, when for a time it will be higher, or just before death, when it may run up to 107° F. The severity of rigors is not looked upon as having any particular meaning, but it is otherwise with their frequency, and their frequent occurrence is only too suggestive of general systemic infection.

RESPIRATIONS.—The respirations are usually below the normal; as few as eleven per minute have been noticed. As the disease advances they become shallow and often sighing, and towards the end they may assume the Cheyne-Stokes character. Sudden stoppage of the respiration while the heart still beats, so peculiar to cerebellar abscess, very rarely occurs, but it may be observed. In case No. 27, in which there was an abscess in the left post-frontal as well as in the temporo-sphenoidal lobe, the respirations ceased before the heart stopped.

EYES—*Pupils.*—The state of the pupils is most important. Certainly it does not assist one in diagnosing cerebral abscess, but it is of decided value in helping one to locate the side on which the abscess is situated; moreover, it speaks aloud as to the stage at which the disease has arrived. If the pupils are equal and respond actively to light and accommodation, we know that the abscess cannot be farther advanced than the

early purulent stage; while if they are unequal, one being widely dilated, the probability is great that the abscess will be found on the side of the one that is the more widely dilated. To this rule the exceptions are few. If both pupils are widely dilated, the abscess is probably large. As the pupils lose their activity and become sluggish or stable, we become strongly impressed with the opinion that the patient is in or near the terminal stage.

When temporo-sphenoidal abscess is fully developed the almost general rule is that the pupil of that side is widely dilated and stable, apart from the presence of third-nerve paralysis, of which dilatation of the pupil would form part. There is very rarely contracted pupil, but it occurred three times in our fifty-nine cases; and when contraction is present, it indicates the disease being in an advanced state and that the pus will be green and offensive; and if with pupillary contraction there is also insensitiveness to light, the patient is in a perilous state.

Photophobia.—This is an occasional symptom, and most frequently seen during the stage of remission; it is not peculiar to abscess in any particular part of the brain or to any stage of the disease.

Diplopia.—Diplopia is also an occasional symptom; it may be noticed in abscess of the temporo-sphenoidal lobe, and is comparatively frequent in abscess of the frontal lobe.

Optic Neuritis.—Optic neuritis means swelling and redness of the optic disc and retina, blurring of the margins of the disc, with the veins first turgid, then

tortuous and swollen, and the arteries constricted. Associated with these conditions are often scattered retinal hæmorrhages, and both the optic neuritis and the hæmorrhages are caused more by the general intracranial pressure than by direct pressure or injury by the abscess. Both are symptoms common to many intracranial diseases. The optic neuritis usually begins to show itself in cerebral abscess when the disease has lasted two or three weeks, but is seldom so severe as to end in blindness; yet in one of the cases in the list a boy had been ill seven weeks with cerebral abscess, and was blind from intense optic neuritis, when an operation relieved all his symptoms except the blindness, which became permanent.

Optic neuritis requires time for its development, and less than ten days is scarcely a measure of that time; therefore it will seldom be exhibited if the abscess runs its course to a fatal termination within that time, and it may not be present at all if the abscess is a small one. In cases that have proceeded for several weeks, or even two months, there may be no signs of optic neuritis; but generally after two weeks of other symptoms there is found some papillitis, or it may be just a slight fulness of the retinal veins, with haziness of the disc margins. On the other hand, optic neuritis may be progressing when all other signs and symptoms, such as headache, vomiting, etc., are in abeyance. The absence of optic neuritis does not negative cerebral abscess, and its presence does not by any means make its diagnosis certain. Its value as a symptom is that, if present only on one side, or if it is more obvious on one

side than the other, that side is probably the site of the lesion. The rule is not absolute, but it is seldom wrong. Optic neuritis may be, and mostly is, present in all conditions in which cerebral hypertension exists; it has also been noticed in anæmic and hysterical girls, and Drs. Barr and Rowan, of Glasgow, have noticed it in the course of otitis media when no intracranial disease was present, but it is notably absent in post-basic meningitis.

HEARING.— The majority of cerebral abscesses originate from suppuration in the middle ear, which has caused erosion of the **tegmen tympani** or **antri**, such suppuration having usually been present for many months and even years; at the same time, it may be stated that a long continuance of otorrhœa is not essential for its production. In case No. 13 there was evidence of abscess three weeks after the aural discharge began. If the otorrhœa has been of long duration the tympanic membrane will be found perforated or perhaps almost obliterated, the ossicles eroded, and loose or absent, particularly the malleus, and the hearing defective. The discharge from the ear usually ceases when cerebral abscess has formed, but such cessation does not always take place; and from now onwards it must be taken that we mean abscess of the temporo-sphenoidal lobe when we say cerebral abscess.

The state of the hearing is of value in the diagnosis of the stage of cerebral abscess, but as coma takes possession of the patient this test cannot be applied. Loss of hearing, partial or absolute, in abscess of the temporal lobe can excite no surprise when we call to mind that the structures which are fundamentally

necessary for hearing may be, and usually are, injured and often destroyed.

In abscess of the temporal lobe there are the same morbid processes at work which produce deafness in cerebellar abscess—*i.e.*, destruction of the conducting auditory apparatus in the middle ear and injury to the auditory nerve in the Fallopian tube; but, in addition, there is destruction of or pressure upon the auditory brain centres in the superior temporal convolution, which govern hearing and appreciation of sound, and cause partial deafness in the opposite ear (mind-deafness).

If we find the **membrana tympani** in a state of acute inflammation—that is, if it has a reddish-brown or copper colour, or individual vessels or bundles of them which radiate from the lower end of the manubrium to the periphery of the membrane (or the whole of the membrane) a uniform bright red colour, or bluish-red if the inflammation is intense; or the membrane denuded of its epidermis looks red, swollen, and spongy, while its fellow of the opposite side is normal in appearance —we have an objective symptom of great value. On whichever side the tympanic membrane is found to be the more highly inflamed, that side will eventually be found to be the side of the abscess. Perhaps at the moment when examination is necessary it is impossible, on account of granulations or small polypi, which require removal before the examination can proceed.

The examination of both tympanic membranes, carried out as a matter of routine, might quickly settle points which sometimes cause much trouble—*e.g.*, a

person may have received a blow or other injury upon one ear, and a superficial abscess near that ear follows; but the intracranial disease which ensues is not located upon that side at all, the force of the blow having been carried by *contre-coup* to an old encysted abscess on the opposite side, around which fresh inflammation arises; or the effect of *contre-coup*, **commotio-cerebri**, originates inflammation where no previous disease existed, and causes abscess. But the medical mind continues to look upon the side which received the blow, and on which there may be an otorrhœa, as the diseased side, the difficulty of diagnosis being increased by the headache having become general. Again, if the membrane have disappeared or be normal, which it may be in childhood, and yet intracranial abscess be present, and pus be observed exuding into the attic, we can only conclude that it comes via the petromastoid canal from the mastoid antrum.

With regard to the hearing itself, the side on which the loss of hearing is the greater is, of course, the diseased side.

The value of the loss of hearing as a diagnostic sign may very likely have to be discounted because the patient was deaf in one or both ears before the onset of this the latest trouble; and if deafness is found to be greater on the side unaffected with the supposed abscess, it requires to be ascertained whether it has or has not been left as a legacy by past disease.

If the patient with cerebral symptoms has suffered from suppuration in both ears, which has resulted in the destruction of both tympanic membranes, and there

is not the guidance of focal symptoms, the side on which the aural discharge is the more offensive is likely to be the site of abscess.

CONVULSIONS.—Convulsions occurred in sixteen of the cases, neither age nor sex having had any particular influence in their development, one of the patients being a man forty-seven years of age and the youngest a girl of ten years. Nor has the cause of the abscess much to do with their occurrence; we find them in cases of traumatic origin, and in those which were secondary to ear troubles, tuberculosis, and syphilis. In a few cases they were general, but in most they were unilateral and Jacksonian in character.

Convulsions are the outward and visible signs of the irritability caused by the pressure upon any part of the cerebral cortex of anything which is normally foreign to the part, such as a new growth, hæmorrhage, or a purulent collection. If the part of the cortex that is irritated contains centres, motor or other, the fit will be preceded by an **aura**, or unusual sensation, as pain, spasm, or paralysis in a limb, or laughing, crying, etc.; and the **aura** refers us directly to the portion of the cortex which is experiencing the pressure. There may be no fit in the ordinary sense, the convulsive movements being confined to one side, one limb, or part of a limb.

If the irritation is in the highest part of the ascending frontal convolution (third frontal), there will follow convulsive movements of the opposite foot; of the middle part of that convolution, similar movements of the hand or arm; and the lowest part of the convolution, similar

movements of the side of the face. When the convulsions are unilateral, they are almost always on the side contralateral to the abscess, but the rule is not absolute; and if the paralysis that follows the convulsion looks like being permanent, it suggests to us that the lesion is a gross one.

Unilateral convulsions, with subsequent weakness or paralysis of the parts convulsed, sometimes constitute the first symptom pointing to cerebral abscess, and if no other symptoms develop, the true nature of the case is only made plain at the necropsy.

It is strange that abscess in identical positions should in one case produce a convulsion and in another none—*e.g.*, we find convulsions distinctly unilateral in one case of abscess of the middle third of the ascending frontal gyrus, and general epileptiform convulsions in another having a similar *locale*. When unilateral convulsions do occur they are of much diagnostic value, and a close acquaintance with the functions of the motor area of the cerebral cortex becomes highly useful. Instead of convulsions we may see a twitching of certain muscles—a phenomenon which will develop into a convulsion if the abscess continues unrelieved.

The value of a unilateral convulsion would be exhibited in a case like the following: A person who has had pneumonia, but in whom after the crisis complete resolution does not follow, as evidenced by cough, sonorous râles, rusty-coloured expectoration, etc., and on bacteriological examination a tuberculous cause for the continuance of symptoms is negatived by the absence of the tubercle bacillus, is a possible subject for cerebral

abscess. If such a one develops severe headache, has a slow pulse, and is subject to one-sided convulsions of the face, arm, and leg, or any of them, abscess in the ascending frontal convolution is very probable. If the medical advisers hold their hands for more pronounced general symptoms, or such localizing symptoms as aphasia or marked paralysis, a fatal mistake may be made. The pulmonary lesion is usually on the side of the brain lesion, but it may not be, and the side of the convulsion, contralateral to that of the lesion, remains the only reliable sign.

Convulsions may be due to shock caused by the bursting of an abscess into the lateral ventricles, or upon the surface of the brain.

Reflexes.—Abnormality of the reflexes is not common in cerebral abscess, and it is chiefly the knee-jerk that deviates from the health standard. If the patellar reflexes are unequally exaggerated, the greater exaggeration is on the side contralateral to the lesion. As a rare event we may see the abdominal reflex diminished or absent on the contralateral side, on which side are also ankle-clonus and the extensor type of plantar reflex, when they occur at all.

Aphasia.—That cerebral defect which prevents a person from understanding the meaning of spoken language is known as **sensory** aphasia, or **word-deafness**—that is, the patient is deaf to the meaning of spoken words, although he is quite able to hear them. It is, when present, an unerring focal symptom of temporo-sphenoidal abscess, and is due to lesion of the posterior half of the superior temporal convolution of

the left temporal lobe in right-handed people, and of the right lobe in left-handed people.

As sensory aphasia can only occur in lesions of a relatively small portion of the temporal lobe, its absence does not negative abscess in the whole of that lobe, as the greater part of it is not concerned with speech; its presence has also this other value—that it places outside the pale of possibility cerebellar abscess except as an associated lesion. A person may have a difficulty in expressing himself, not because his speech-centres, situated in a certain area of the cerebral cortex, are being interfered with, but because his mental faculties are clouded through the toxic influence and general intracranial pressure of the second and third stages of the disease. It is important to distinguish between the varieties of aphasia, and this point will receive attention when the differential diagnosis of abscess of the brain is being considered.

When aphasia continues after an epidural abscess over the temporal lobe has been evacuated, and the pulse continues abnormally slow and the temperature subnormal, there is good reason for concluding that there is an intracerebral abscess, notwithstanding the well-known fact that aphasia and paralysis may not show signs of improvement quickly.

Agraphia (the inability to recall or arrange written characters so as to form a word or sentence) and **alexia** (the inability to recognize written or printed characters, or to read aloud correctly) refer a lesion to the right half of the cortical visual centre in the occipital lobe, the visual word-centre in the angular gyrus, or, what is

more probable, to the fibres connecting the latter with the motor speech-centre; they occurred in one case of temporo-sphenoidal abscess, the first-named *locale* being ruled out by the otitic origin of the trouble and the other symptoms.

Taste and Smell.—These senses are usually normal, the seat of suppuration being for the most part distant from that which is concerned with taste and smell. Dr. Risien Russell says that fits, beginning with an **aura**, relating to these special senses, may occur, and be followed by the peculiar condition known as the dreamy state.

Paralysis.—During the progress of an abscess in the brain paralysis may occur immediately after a convulsion, or it may develop more or less gradually, according as intracranial pressure or irritation increases rapidly or slowly. The paralysis of the limbs in cerebral abscess, whether monoplegia or hemiplegia, is on the side contralateral to the abscess, and spastic rather than flaccid.

Unilateral paralysis of the extremities was present in rather less than half the cases in our list—to be precise, twenty-nine times in the fifty-nine cases. From an early stage of temporal lobe abscess the grasping power of the hand on the healthy side is very much less, as shown by the dynamometer, than that of the affected side; but actual paralysis is one of the conclusive signs, with sometimes the paralyzed muscles rigid, of the terminal stage.

The paralysis may be hemiplegic, or affect only certain groups of muscles in the arm or leg, or both, a

portion of the face or the whole of one side of the face.

As the centres for arm, leg, and face are in such near proximity, it is little wonder that paralyses of these parts occur so often in concert, paralysis of one of them suggesting a circumscribed collection, and of the three together a relatively large one. The abscess may not be immediately under the centres; it may be deep in the white matter or in the neighbouring part of the cortex. If the paralysis is irritative only, caused by an associated encephalitis, it will be recovered from; if absolute destruction of the motor centres has taken place, it will be irrecoverable.

We have spoken of the face, arm, and leg being paralyzed on the healthy side, but it is of importance to know which of these parts was the first, and the worst, paralyzed, as pressure upon the internal capsule and crus cerebri produces similar paralysis, but in a different order. The two last will be considered under Differential Diagnosis, and it will be sufficient at present to say that in the pressure of a temporo-sphenoidal abscess upon the sensori-motor convolutions of the Rolandic area the face suffers first and most, the arm next, and the leg last.

Cranial Nerves.—Ptosis is common, on account of the nearness of the third nerve to the crus cerebri, and with it the other indications of third-nerve paralysis, which are, when complete, loss of inward, upward and downward movements of the eyeball, on the same side as the drooping eyelid; loss of accommodation, partial mydriasis, and well-marked **divergent** strabismus, from

the unopposed action of the external rectus; and crossed diplopia. The paralysis, however, is most often incomplete.

The facial paralysis which may occur is (*a*) cortical and rare, or (*b*) due to injury of the nerve in its passage through the Fallopian aqueduct—the more usual. In the former it is incomplete and on the side **opposite** the lesion, and in the latter it is complete and, of course, on the homolateral side.

Diagnosis.—The diagnosis of cerebral abscess depends upon focal symptoms developing after general ones have been present for some time. The most important of the former class are aphasia and other defects of the cerebral speech-centres, and hemiplegia on the side opposite the supposed abscess.

Headache, vomiting, drowsiness, irritability, etc., are common to all acute intracranial diseases, while optic neuritis, the condition of the pupils, and the state of the reflexes, refer us to the side on which the abscess is, rather than to the fact that there is abscess.

Post-Operation.—The only possible treatment is operation, the management of which does not form part of this work. Unless the patient is actually moribund at the time when the pus of an intracranial abscess is liberated—that is, in an uncomplicated case—good effect is at once noticed, even if the patient die later on. As pus flows, the pulse may be found to rise, although it may slow again in two or three hours. If he recovers consciousness, he may be bright and intelligent. Sometimes the various forms of paralysis may disappear immediately after the operation, when they

have been caused by pressure only; at other times in a few days. Speech returns to its former quality, and in the great majority of cases sight is restored in a few weeks or a month or two. Unfortunately, this much-to-be-desired ending is not invariable. The patient may be subject to certain disabilities on account of the dura mater having become adherent to the skull, causing him to faint on sudden movement or when brought into contact with great heat; or a hernia cerebri may develop, and cause death by septic poisoning; or the facial paralysis, the ptosis, or the hemiplegia may not disappear.

Continued loss of hearing after the radical mastoid operation suggests that the nerve has been destroyed by disease or removed by the surgeon in clearing out the tympanum.

Cerebellar Abscess.

If we found cerebral abscess or abscesses—for it must always be remembered that there may be more than one—occurring more frequently in males than females, it must now be stated that cerebellar abscesses are more likely to do so.

Ætiology.—The remarks that have been made as to the connection of otitis media with cerebral abscess, especially when it follows measles or scarlet fever, apply still more forcibly to cerebellar abscess. Some writers insist that all cases of the latter are due to ear troubles, but such conclusion is not quite justifiable, as violence and syphilis provide occasional cases; but there is this difference in the ætiology of the two great varieties of

brain abscess: otitic cerebral abscess usually arises directly from suppuration in the tympanum, the temporo-sphenoidal lobe lying upon its roof; while otitic cerebellar abscess, the cerebellar hemisphere lying beneath the tentorium, arises most frequently from suppuration in the mastoid cells, from continuity of the suppurative process along the mucous membrane which passes into the cells from the tympanum; but there are exceptions to the rule.

Out of the thirty-nine cases which the author has collected, thirty-seven were known to have originated from diseases of the ear; of the two remaining cases, one was due to the breaking down of a gumma, and the cause of the other was not stated: it may or may not have been the ear. It is thus manifest that violence can only play an infinitely smaller part than it does in the causation of cerebral abscess; indeed, some writers seriously question whether it plays any part. Practically, cerebellar abscess is primarily due to suppurative otitis media, acute or chronic, the greater number of cases being from the latter, even if there is no discharge or has been none for a long time previously.

Acute and chronic abscesses arise in a widely divergent way. The acute are consequent to phlebitis or thrombo-phlebitis of the lateral sinus, or to an extradural abscess in the posterior cranial fossa, and to the poison carried by the perivascular lymph channels; when suppurative labyrinthitis is present, it is secondary to abscess. The chronic are due to labyrinthine suppuration, started by cholesteatomata or osteomyelitis of that portion of bone which is situated between the lateral

sinus and the labyrinth and bounded above by the superior petrosal sinus.

The cholesteatomata, originally consisting of dried shed epidermis, which may have accumulated in the antrum and tympanum, may not be sufficiently septic to originate *per se* any serious disease; but they form a fitting nursery for septic foci when a more powerful micro-organism reaches them, and this is often supplied by scarlet fever and measles. It obtains entrance by way of the Eustachian tube to the tympanum, and thence advances to the mastoid cells. Rapid development follows, and deadly foci are carried along existing, and perhaps also new, channels, and pyogenic diseases follow, of which cerebellar abscess is one. Further, the fact that the floor of the antrum is upon a lower level than that of the tympanum, and cholesteatomata in the external auditory meatus, prevent the passage of pus and help on the inflammation and decay of bone contiguous to the imprisoned pus.

The pathology of cerebellar abscess runs on the same lines as the cerebral abscess; therefore the formation of red softening due to an infective agency, its breaking down into abscess, its probable encapsulation, and the structure of the capsule, need not be again commented upon.

Too great an emphasis cannot be placed upon the fact that cerebellar abscess—at any rate, when that disease is consequent upon aural troubles—only sometimes constitutes the whole trouble; it often forms only one item, albeit not the least important one, in a group of pathological states, cephalic and at times non-

cephalic, any of which, being unrelieved by operation, will cause death; those infinitely rare cases which open through a carious opening in the skull or the external auditory meatus by the agency of a ruptured tympanum being practically valueless as possibilities.

The presence of one or more of these associated diseases may profoundly modify or altogether destroy the symptoms which might be present if the case were uncomplicated.

The cephalic complications may include pus or fœtid cholesteatomata in the tympanic attic or mastoid antrum, or both; caries of bone or osteo-myelitis; extradural abscess, which is really a circumscribed purulent meningitis, due to the gluing together of the surrounding inflamed meninges; abscess of the temporal lobe; suppurative labyrinthitis, which may be distinct or communicate by a dural fistula with the abscess; phlebitis or thrombosis of the cerebral sinuses, the sigmoid being the one most frequently affected, then the remaining part of the lateral, and less commonly the petrosal, transverse or cavernous sinuses, in any of which the clot may break down and produce pyæmia; or there may be inflammation about a cerebral sinus—perisinusitis—the vessel lying in a thin ichor. The wall of such sinus may be red, indicating inflammation only, or it may have undergone degenerative changes, and be green or even gangrenous.

The most frequent non-cephalic changes are thrombosis of the external jugular vein, abscess of the spleen, and pulmonary and retropharyngeal abscesses.

The most frequent path of conduction from suppu-

rative labyrinthitis is by the aquæductus vestibuli. Neumann gives, in addition, the internal auditory meatus, porus acusticus internus, aquæductus cochleæ, and a fistula in a semicircular canal.

The earliest age in our list at which cerebellar abscess occurred was eight years, and the latest fifty years, early adolescence claiming the largest number of victims.

The vermis is only rarely affected, but either lateral lobe may be the site of abscess. In our cases the right lobe was the more frequently affected, in the proportion of 22 to 13. Other writers give the preponderance to the left side.

For cerebellar symptoms to be excited it is not necessary that the abscess should be intracerebellar; pressure upon the organ from without will just as readily produce them as pressure from within. The abscess may be extradural, between the pia mater and cerebellum, between the cerebellum and tentorium, or supratentorial, as occurred in one of the listed cases. The unassigned causes in the appended list may have been ear troubles, but they were not given.

No.	Cause.	Site.	Age in Years.		Associated Disease.
			M.	F.	
1	Otorrhœa, after scarlet fever	—	—	14	—
2	Otorrhœa -	—	26	—	—
3	,, -	R. lobe	29	—	—
4	,, -	R. ,,	42	—	—
5	,, -	R. ,,	14	—	—
6	,, -	R. ,,	—	8	—
7	Otorrhœa, after scarlet fever	R. ,,	14	—	—
8	Otorrhœa, after scarlet fever	R. ,,	14	—	—

No.	Cause.	Site.	Age in Years.		Associated Disease.
			M.	F.	
9	Otorrhœa, double	R lobe	—	—	—
10	Otorrhœa -	R. „	31	—	—
11	„ -	R. „	22	—	—
12	Otorrhœa, after measles	R. „	14	—	—
13	Otorrhœa -	R. „	—	—	—
14	„ -	R. „	—	—	Aural polypus.
15	„ -	R. „	19	—	—
16	„ -	R. „	14	—	—
17	„ -	R. „	22	—	Petrous bone rough, superior petrosal sinus thrombosis.
18	„ -	R. „	15	—	—
19	„ -	R. „	28	—	—
20	Syphilis - -	R. „	46	—	—
21	No cause known	R. „	—	—	—
22	Otorrhœa -	L. „	14	—	—
23	„ -	L. „	42	—	—
24	„ -	L. „	23	—	—
25	„ -	L. „	17	—	—
26	Otorrhœa, double	L. „	—	—	Infective lateral sinus thrombosis.
27	Otorrhœa -	L. „	15	—	—
28	„ -	L. „	—	—	—
29	„ -	L. „	23	—	—
30	„ -	L. „	—	—	—
31	„ -	L. „	26	—	—
32	„ -	L. „	50	—	—
33	„ -	L. „	21	—	—
34	„ -	L. „	13	—	—
35	Otorrhœa, double	—	—	—	Sinus thrombosis.
36	„ „	—	13	—	—
37	Otorrhœa -	—	—	44	—
38	„ -	Extra-cerebellar	—	12	—
39	„ -	—	—	—	Aural polypus.

Symptoms.—Abscess of the cerebellum may run a latent course, the patient dying suddenly without having exhibited any symptoms, or only those of rupture into the ventricles, or only the general symptoms of intracranial abscess may be present, and

no localizing ones. The symptoms of the febrile stage agree generally with those of cerebral abscess, except as to the site of the pain; but with the advent of the second stage differentiation, as a rule, becomes possible.

Cerebration is slow, as in cerebral abscess, but speech is more hesitating (dysarthria) and difficult (anarthria), which, with the difficulty there may be in swallowing, are due to pressure of the abscess upon the pons. There may be also incontinence or retention of urine, and the patient emaciates more rapidly.

It has been noticed elsewhere (tumours of cerebellum) that albuminuria is occasionally present in cases of cerebellar tumours, and disappears with returning health; the same might have been said of glycosuria and intense thirst; and all these hold good with regard to abscess, both in the matter of appearance and disappearance.

Convulsions are rare. There are two points of time when they are likely to occur: when the encephalitis is breaking down into abscess, and shortly before death, when they are suggestive of rupture, particularly if associated symptoms are exaggerated. If operation has relieved symptoms, and in the course of a day or two convulsions recur, it is a sign that the drainage is inadequate, or that there is another and distinct abscess.

The patient may fear the light as much in cerebellar abscess as in meningitis, so photophobia cannot be relied upon as a distinguishing mark between the two.

Headache.—Headache, perhaps starting as earache, may be dull and heavy, or severe and of a boring character, and liable to occur in paroxysms of great

intensity, but becomes mitigated as sensibility is deadened. The site of the pain is usually persistently occipital, and may be localized upon one side of the occiput; the next prominent *locale* is the forehead, or it may be general over one side of the head, and is on rare occasions only experienced at the vertex. The striking connection of frontal headache with cerebellar disease may be occasionally noticed by starting frontal pain on passing fresh drainage-tubes into the abscess cavity.

Tenderness upon pressure or percussion of the occiput is often complained of, and the skin over the occipital area may be highly sensitive; but these symptoms are more suggestive of the abscess being extradural than intracerebellar, and their value as cerebellar symptoms is discounted by the fact that they are observed in the meningitides, lateral sinus thrombosis, and in caries of the occipital condyles and of the atlas and axis. Mr. Hilton, in 'Rest and Pain,' draws marked attention to the latter cases.

Rigidity of the cervical muscles and pain down the neck are not unusual, and retraction of the head is frequent—more so than in cerebral abscess, but less often and to a much less degree than may be seen in cerebro-spinal meningitis. It is said to be due to extension of the intracranial pressure to the posterior roots of the upper cervical nerves.

Vomiting.—Vomiting, with or without rigors, or retching without vomiting and apart from the taking of food, and associated with a feeling of dizziness, is almost always present, and may appear for the first

time or be worse as the second stage of the disease supervenes and develops; and the vertigo may be often started by raising the patient to the sitting posture. Vomiting and vertigo may also be produced by other conditions, notably by anything which is likely to start vestibular irritation, as by the pressure of hard wax upon the tympanum; indeed, both symptoms may be produced artificially if one inadvertently continues to syringe the ear after all cerumen has been removed, or where there is really no wax to remove. Nausea may be very unpleasantly present in the second stage, when vomiting is altogether absent, and the idea of food is more loathsome to a patient than in the case of a cerebral abscess of the same duration.

The tongue is, as a rule, thickly coated and furred when there is vomiting, and red and glazed when only nausea is present; but in either case the sickly odour of the breath is just as offensively obvious as in cerebral abscess—possibly more so. We are speaking of its condition after the inflammatory stage of the disease has passed. The bowels are likely to be constipated until late in the terminal stage. Vomiting is certainly more frequent in cerebellar than cerebral abscess, due, it is said, to the more frequent association of lateral sinus thrombosis with the former.

Temperature.—There is really nothing *diagnostic* of cerebellar abscess in the state of the temperature; it is sometimes normal when there is obviously serious disease present, but the usual temperature in the fully developed uncomplicated disease is a subnormal one, and the lower it is the greater is the danger. On the

other hand, we may have a febrile temperature of five, six, or seven degrees; but in such cases the probability of some other disease, as sinus thrombosis or meningitis, being present is great. If the associated disease is meningitis, its nature is explained by the behaviour of the temperature after evacuation of the abscess; if the temperature becomes normal, the meningitis is serous; if it continues febrile, it is the diffuse purulent. A rigor may be one of the initial symptoms.

PULSE.—The pulse is quick at the onset of the disease, but when the second stage is reached it is almost invariably slow; 60 to 65 beats per minute is the rule, but there may be only 50, 40, or even 30, and possibly weak and intermittent, although no heart trouble exists. A slow pulse is even more suggestive of cerebellar abscess than a subnormal temperature, and if it is quicker than 80 or 90 it agrees with the febrile temperature in bespeaking the probability of some other intracranial disease being present. An arrhythmic pulse is common in the earlier stages, but it is also present in other acute diseases of the brain.

RESPIRATION.—Patients may breathe during the greater part of their illness in quite a normal way, but in most cases the respirations are fewer per minute than is natural, and this respiratory slowness is noticed oftener than in cerebral abscess. Towards the end of the illness, and before coma becomes deep, patients may exhibit the Cheyne-Stokes manner of breathing. Sudden stoppage of the respiration while the heart continues to beat is the great dread, and may be the

cause of death in many patients who die suddenly while suffering from cerebellar abscess.

This catastrophe has occurred many times while operation was in progress. If the surgeon thinks that the condition is due to the anæsthetic, when one is being given, his patient dies; if, on the other hand, he rightly grasps the situation and hastens his procedures, while others carry out artificial respiration, he may have the pleasure of seeing natural breathing return as the abscess is emptied. This dangerous accident is caused, as is also the case in unnaturally slow breathing, by increased pressure upon, or inflammatory œdema around, the vital centres in the bulb, or by both causes. One would suppose that pressure is the more powerful agent, as emptying the abscess is often coincident with the return of normal breathing. This condition occurs occasionally in the case of cerebellar tumours, and is a by no means uncommon occurrence in abscess, as the following recorded cases show:

Case 1.—Mastoidectomy had been carried out without result, when the patient suddenly ceased breathing, the pulse being good and regular. The cerebellum was opened during artificial respiration, pus was evacuated, with rapid improvement of the breathing. Good recovery.

Case 2.—Child, eight years old, suffered from abscess of the cerebellum. Operation was far advanced when the face became deeply congested and breathing ceased, the pulse at the wrist being good. The heart only ceased to beat six hours after the breathing had stopped.

Case 3.—Boy, seventeen years old, suffered from cerebellar abscess. The right lobe had been operated upon without result, when the breathing ceased, the pulse being still strong. The left lobe was operated upon during artificial respiration, and spontaneous breathing began the moment pus oozed out.

Case 4.—A man, forty-four years of age, was on the point of being operated upon for relief of a supposed cerebellar abscess, when he suddenly stopped breathing. The skull was quickly opened over the cerebellum, and breathing was rapidly re-established as cerebro-spinal fluid escaped. There was no abscess.

CONVULSIONS are uncommon in cerebellar disease; they occurred three times in eighty cases of tumour and only once in the thirty-nine cases of cerebellar abscess, and in that the patient was unconscious. Mr. Balance, in 'The Surgery of the Brain,' quotes a case in which the patient remained conscious.

FOCAL SYMPTOMS—*Attitude and Rigidity of the Nuchal Muscles.*—If the patient is seen in the early stage of the disease, while he can yet walk or stand, it will very likely be noticed that he keeps his head rigid and fixed, possibly supporting it with both hands, and that then, and later, he may have his head drawn back and the face towards the side of the otorrhœa, or, if that is on both sides, towards the side of the earache.

The decubitus is that of lying curled up upon the healthy side, with the legs flexed, or upon the back, with the legs extended and toes pointed, the former being common in the early stages and the latter when coma supervenes.

The Pupils.—Until late in the disease the pupils are dilated, and usually equally so; if unequal, the larger one is mostly on the affected side. The sensitiveness of the pupil to light is a hopeful sign, as sluggishness is not; absolute inactivity is bad, and contracted pupils are an almost certain forecast of approaching death.

Optic Neuritis.—Optic neuritis is more frequent in cerebellar than cerebral abscess—at any rate, that variety known as choked disc, in which there is œdema of the disc and venous engorgement, is said to be. It is not noticed early in the disease, and in some cases is altogether absent (it was absent six times in our thirty-nine cases); but when once it appears, it advances rapidly until a state of intense neuritis may be present. A week or two may produce as intense optic neuritis as several months may do in the case of tumours, and although there may be no neuritis there may be amaurosis, the patient complaining that he cannot see, and, as in cerebral abscess, the intensity of neural inflammation is no indication of the size of the abscess. Optic neuritis first appears on the affected side, or if it appears simultaneously on both sides, the eye on the affected side may be worse than the other. The appearance of optic neuritis first on the affected side is the generally accepted rule, and was the condition in all of the cases in our list in which optic neuritis was present at all; but there are undoubted exceptions, and its value as a localizing sign is therefore not absolute. Optic neuritis may be present when only mastoiditis occurs. Retinal hæmorrhages are suggestive of the terminal stage. Pressure of the abscess upon the occipital lobe may

bring about some restriction of the visual field, but it will not amount to hemianopsia; when there is such limitation, it is on the affected side, or worse on that side.

Attitude and optic neuritis have been placed with the localizing symptoms, as being to some extent indicative of the side of the lesion, but most writers include them under general symptoms.

Ataxia.—The loss of power to cause one set of muscular movements to act in agreement with others, or muscular inco-ordination, and particularly hemiataxia, is, along with hemiparesis, hemianæsthesia, and other focal symptoms, liable to be exhibited during the course of the second stage.

The characteristic disturbances of equilibrium, such as staggering gait, etc., are either caused by abscess in the middle lobe, which is very rare, or by pressure exerted upon the middle lobe by lesions in the lateral lobes, which may be destroyed by tumour or abscess, or removed by experiment on the lower animals, without the power of walking correctly being affected. The peculiar staggering walk or reel, as of a drunken man or of a person on the deck of a ship when the sea is rough, is very suggestive of a cerebellar lesion. This gait, with the body forward and feet wide apart, is to be distinguished from that of tabetic ataxia, in which the body is held stiffly back and the feet occupy the forward position.

If the patient is unable to walk the ataxic state may be recognized by the erratic method of picking up a pin or other small article. If ataxia is present, there is also

likely to be a liability to fall to one or other side, that side being the diseased one. This is particularly the case when the eyes are closed, but this liability to fall may be lost if the patient stands with his legs wide apart, or because, being aware of the tendency, he overcorrects it, and falls to the opposite side.

It must be remembered that ataxia is an occasional symptom in lesions of the prefrontal lobe, and abscess there has often been thought to be cerebellar; but the cerebellum has rarely been operated upon, and the lesion afterwards found located in the frontal lobe.

Hemiataxia, or motor ataxia of the limbs of one side, when it occurs in cerebellar abscess, is of twofold value, as it not only indicates the cerebellum as the site, but it points directly to the abscess being situated upon the same side.

As a test for defective inco-ordination, diadokokinesis is very valuable, and consists in rapidly alternating movements of arm and forearm, these movements being irregular and inco-ordinated on the side of a cerebellar lesion; but the test is only responsive in some cases of cerebellar disease.

Paralysis.—Absolute paralysis of the arms or legs is rare, but a hemiparesis of the limbs of the **affected** side, or of one of them—and the arm is the limb most often affected—is not uncommon, and is equally valuable with hemiataxia and hemianæsthesia as a localizing sign; and the fact cannot be adverted to too often that hemiparesis on the **affected** side is a distinctly cerebellar sign and hemiparesis on the **sound** side is a distinctly cerebral sign. In the place of paresis there may be

tremors or clonic convulsions in the homolateral limbs, and in the terminal stage flaccidity of the paralyzed or partly paralyzed muscles is their usual state; but neither paralysis nor paresis is always present, and when there is neither of these a careful comparison of opposite limbs may reveal the fact that on the homolateral side there is a weakness, or hypotonus, not seen on the other side.

The cranial-nerve paralysis most often seen is that of the seventh nerve, which may be produced in any one of these ways, the most frequent cause being injury to the nerve during its passage through the aqueduct of Fallopius. The other causes are by pressure of the abscess upon the pons Varolii, when there may be a bilateral facial palsy or a crossed paralysis of the face on the homolateral and of the limbs on the contralateral side; or by pressure upon the nerve at its point of entry into the internal auditory meatus, when you would also observe paralysis of the soft palate on the homolateral side.

Facial paralysis may be the earliest symptom of cerebellar abscess, and this fact should be kept in mind when it suddenly becomes apparent during a chronic otorrhœa. In the place of paralysis, the muscles supplied by this nerve may exhibit twitching or tremors.

On rare occasions the sterno-cleido-mastoid may show rhythmical contractions, pointing to the nervus accessorius (eleventh) being involved (Neumann).

The next most frequent cranial nerve paralysis is that of the sixth nerve, the abducens, and we see a convergent squint on the affected side. The trouble which

affects the abducens may not amount to paralysis, but only to a disability in the external rectus to draw its eye conjugately with the eye of the sound side towards the side of the lesion. There may be conjugate deviation of the eyes, and when present the deviation is invariably towards the diseased side.

Paralysis of the third nerve is very rare, and is shown by a drooping of the upper eyelid on the affected side; but it is more often seen in temporal lobe abscess.

The sense of taste may be lost on the affected side.

Reflexes.—The reflexes are only occasionally abnormal. The knee-jerk is liable to be exaggerated on the affected side; it may be diminished in subacute cases and lost in chronic ones.

The corneal reflex may be lost in the terminal stage.

Vertigo.—Quite apart from vomiting, dizziness is often present, along with a feeling of faintness. If the patient is recumbent, he may fall backwards or forwards on trying to sit up; if he is standing or walking, he has vertiginous attacks when attempting to turn, perhaps falling to one side, which conveys the direct suggestion that the lesion is on that side. Many patients exhibit the Romberg symptom—that is, falling to one side, usually the side of the lesion—when trying to stand or walk with the eyes closed.

Nystagmus (from *νυστάζω*, I nod).—Nystagmus consists of involuntary and frequent rhythmical movements of the eyeball, produced by alternate contractions of the opposing muscles. The movements may be horizontal (lateral), rotatory, or vertical; the condition is usually bilateral, and becomes worse when the patient

has his eyes attentively engaged. Its causes are many and varied, but it must be considered sufficient for our purpose to say that it may be present in tumour or abscess of, or hæmorrhage into, the cerebellum, optic thalamus, and pons Varolii, and in vestibular labyrinthitis. Vestibular nystagmus depends upon irritation of some part of the vestibular tract, but any stimulation of the semicircular canals causes movements of the eyeball corresponding to the plane of canal injured.

Nystagmus, when concomitant with the general symptoms of intracranial abscess, may be looked upon as pointing to the cerebellum, and it is present in the majority of cases of cerebellar abscess. Like hemiparesis and hemiataxia, it has a double value—it not only suggests the cerebellum as the site of the lesion, but also indicates the affected hemisphere, as the more rapid movements are towards the affected side.

Hearing.—If disease of the middle ear, originating from a zymotic disease, a polypus, or cholesteatomata in the external auditory canal, be accepted as the cause of cerebellar abscess, no surprise will be occasioned on hearing that by the time cerebellar abscess is developed the primal disease may be found to have caused wholesale destruction of the auditory apparatus. The tympanum may on rare occasions be intact, but it is usually badly perforated, and may, indeed, have completely disappeared. One or more of the ossicles of the middle ear—the incus and malleus more often than the stapes —may have become necrosed, as also may have the labyrinth, cochlea, and semicircular canals, and labyrinthine suppuration be in progress.

More or less deafness is certain—and, as a matter of fact, it is an early and constant symptom—at first to aerial and later to bone conduction, at any rate on the affected side; but tinnitus, so often present and harassing in the case of tumours, is seldom met with.

If the hearing is not tested in an early stage of the disease by the watch or tuning-fork, it may be found impossible to do so at a later stage, when the mind has become clouded. Destruction of the semicircular canals does not destroy hearing, but causes a person to feel dizzy and to be unable to balance himself. If the horizontal canal only is in fault, the patient turns his head horizontally when he moves; if the posterior or superior, he may fall backwards or forwards, as the case may be.

Diagnosis.—If it has been concluded that the trouble is intracranial abscess, the question arises, Which variety? as many symptoms are common to all acute diseases of the brain, and the difficulty is much increased if the patient has come under notice late in the disease, and an intelligent history of the case is not possible. Before we can arrive at the diagnosis of cerebellar abscess, the possibility of cerebral abscess must first, if possible, be eliminated, bearing in mind the fact that the two may coexist; then it will have to be considered whether labyrinthine suppuration be present, either by itself or in conjunction with cerebellar abscess. Further, are the symptoms present those of sinus thrombosis, or is the latter also an associated trouble?

If double otitis media be present, it may occasionally, unless the focal symptoms, such as the direction of

nystagmus, be very definite, be a matter of difficulty to say which side of the cerebellum the abscess is upon; but the site of the pain and unilateral optic neuritis, if occurring on the same side, would be very helpful in case distinct focal signs were absent.

Prognosis.—Cerebellar abscess is a disease always dangerous to life; without operation, practically the whole number of sufferers die. We hear of persons dying because a diagnosis could not be made; we hear of them dying after operation has been determined upon, in the short interval that must often elapse between operative intention and act. Even in an uncomplicated case, before the symptoms of the terminal stage have become apparent, it is unwise to assure recovery after operation, and when any of the several complications are present the condition is in the highest degree serious, and only a very guarded opinion is possible. Thirty-five cases in our list were operated upon, with eighteen deaths, or a little over 50 per cent.

The size of the abscess is material: naturally, the less the destruction of brain tissue, the greater the chance of life; and there may be more than one abscess, or the abscess may be single and communicate with a large pocket by a small opening.

Diffuse purulent meningitis is perhaps the most frequent cause of death; next comes sudden stoppage of respiration during operation, or a few hours or days afterwards, autopsy revealing the fact that a second abscess has not been reached, or the drainage of the abscess has been defective through the kinking of the

drainage-tube; hence the necessity for using rigid ones.

The patient often rallies for a few hours and then dies suddenly; if he is raised to take food, he may fall back dead. He may die in convulsions, and although the abscess has been thoroughly emptied, the toxæmia of the third stage cannot be thrown off, or the sloughy condition of that portion of the cerebellum which remains keeps up the toxæmia. If the toxæmic stage has been reached, a dulled intellect and an increasing desire for sleep denote that little time is to be lost if the life of the patient is to be saved.

Treatment.—The only possible treatment is evacuation, and as the author is not an operator, he is only in a position to offer general remarks upon the subject. An operation for abscess, cerebral or cerebellar, will, so far as it goes, benefit purulent meningitis; therefore the latter does not bar operative procedure, but general pyæmia certainly does. The chisel and mallet may well give way to bone forceps and circular saw, as the violence of the former may do incalculable mischief, causing rupture of the abscess capsule and flooding the ventricles or surface of the brain with fœtid pus, and when on the verge of success make the case hopeless. Such, indeed, have been the writer's thoughts on seeing the above tools—they can scarcely be termed instruments—in use.

Another point—perhaps unnecessary to urge at the present day, but certainly vital—is that the skull should only be opened over the supposed site of abscess in cases of non-otitic origin, or of extreme urgency brought

about by sudden stoppage of the respiration; and that in all otitic cases it is unreasonable to attempt to cure the disease except by eradicating it at its starting-point and beginning the operation from the point of infection in the middle ear; and as drainage-tubes are a necessity, they should be rigid, and measures taken to prevent the holes in the tubes from becoming blocked.

When focal symptoms are unquestionably cerebellar, and no pus is found on careful and repeated exploration, the exploring needle should be pushed through the tentorium cerebelli, as the abscess may be supra-tentorial.

The Post-Operation State.—Headache is often at once relieved, and so is the vomiting if it has persisted, and it is almost the universal rule for the optic discs and retinæ to return almost to their normal state and the eyesight to be regained—*i.e.*, the neural inflammation has not lasted long enough to produce optic atrophy.

The convergent squint and drooping eyelid may remain as reminders of the past storm.

The deafness will, in all probability, continue. Nystagmus is very likely to remain for an indefinite time, the only change being that the fine and quick oscillations will have become coarse and slow.

CHAPTER IV

DIFFERENTIAL DIAGNOSIS

WHEN a person exhibits symptoms of cerebral meningitis, the question arises as to the variety of the meningitis, or if the meningitis is in association with abscess of the brain or sinus thrombosis. If the patient is under three years of age, and cerebral symptoms, ushered in by persistent vomiting and convulsions, appear suddenly and run a very rapid course to coma and death, the differentiation between acute meningitis and meningeal hæmorrhage may be impossible; in such circumstances it may be well to remember that the temperature may be febrile in the former and afebrile in the latter.

If the patient is known to be suffering from any form of tuberculous disease—glands, bones or joints, as well as the lungs—we are justified in suspecting a tuberculous state of the meninges; but 'tubercle in the choroid may be a mark of a general tuberculosis, and is not diagnostic of tubercular meningitis' (James Taylor), and there may be no tubercles on the choroid.

In Calmette's ophthalmo-reaction of applying a weak solution of old tuberculin to the conjunctiva, which

produces a mild conjunctivitis in a few hours, we have an unerring test of tuberculosis; but it is occasionally dangerous to the eyesight, and its application is consequently undesirable. The cuti-reaction of Von Pirquet is not dangerous, and is only a little longer in producing a result. It is carried out by vaccinating with a sterilized lancet, with a 25 per cent. solution of old tuberculin in 0·5 per cent. of carbolic acid, the tuberculous reaction being the formation of a papule within twenty-four hours.

In lumbar puncture and examination of the cerebro-spinal fluid we have the most definite and precise information that it is as yet possible to obtain, as it may reveal to us Koch's tubercle bacillus, the form of cells that is in excess, the presence or absence of albumin or blood, and how the latter presents itself in the vessel into which the fluid is drawn.

Normal fluid is clear, like water, contains only a trace of albumin in the form of serum globulin, and only mononuclear leucocytes, and the results of culture and animal experiments are negative. The diagnostic value of the clearness is lessened by the fact that the foramen of Magendie may be occluded, and the pus of an encapsulated abscess may be free from bacteria.

Meningitis of some kind is present if the albumin is increased and reducing agent decreased; the fluid is at high pressure and opaque, and the polymorphonuclears are in excess.

In **suppurative meningitis** the fluid is purulent and at high pressure; there is an excess of leucocytes in the blood, and polymorphonuclears in the cerebro-spinal

fluid; there will be found in the fluid streptococci and other micro-organisms.

In **epidemic** and **sporadic** cerebro-spinal meningitis the fluid is at high pressure and usually cloudy, but may be clear or bloody; polymorphonuclears are in excess, as they also are in the blood, but there may be also in the fluid a few mononuclears, red blood-corpuscles, and endothelial cells, the mononuclears being in great numbers if the disease is running a slow course. The meningococcus or pneumococcus may be found in the fluid.

In **serous meningitis** the fluid is clear, and the amount of albumin normal.

In **cerebral abscess** and **sinus thrombosis** the fluid is at high pressure, clear and without polymorphonuclears, if there is no associated meningitis; there may be a mononuclear leucocytosis, but it is not so excessive as in tubercular meningitis. In cerebral abscess there is a high leucocytosis in the blood.

In **tubercular meningitis** there is a clear coagulable fluid, containing 1 gramme (15·4 grains) of albumin in each litre (35·196 fluid ounces), with a large percentage —it may be 90 per cent.—of mononuclear cells. A similar lymphocytosis occurs in syphilitic nervous diseases, such as tabes dorsalis, also in general paralysis of the insane, now looked upon by many as syphilitic in origin. The reducing agent is decreased in the early stages of the disease and absent later. The presence of Koch's tubercle bacillus is conclusive of the disease being tubercular meningitis, but the bacillus may easily be overlooked if the centrifugalization is incomplete or

the fluid contains blood; and it may be mentioned that if there is blood in the cerebro-spinal fluid of tubercular meningitis, 'it is suspended like a fine cobweb from the surface of the fluid, and does not adhere to the sides of the vessel in opaque friable flakes.'[1] The tubercle bacillus has on many occasions not been isolated, and the post-mortem examination has afterwards proved the disease to have been tubercular meningitis.

In acute suppurative meningitis we have very often the facts to guide us that a person, child, or adult is, or has been, suffering from an otorrhœa, or nasal discharge, or acute febrile disease, and the symptoms follow on to coma with much greater rapidity than in tubercular meningitis. Further, the onset is more sudden, the initial fever is higher, the head retraction more marked, the cerebro-spinal fluid is purulent, there is a polymorphonuclear leucocytosis, Koch's bacillus is absent, and the streptococcus, staphylococcus, and other micro-organisms may be present. If the usual symptoms of cerebral meningitis be in evidence, and the retraction of the head is extreme, and there are violent stabbing or shooting pains down the middle of the back, there is evidence sufficient for the diagnosis of suppurative cerebro-spinal meningitis, whether the primal cause of the disease was suppurative otitis media, suppuration in the accessory cavities of the nose, or influenza.

Differentiation between hysteria and cerebral meningitis may be extremely difficult, especially when the hysteria is in association with an infectious disease. Apart from the light which lumbar puncture would

[1] Stirling Landon, *Lancet*, April 16, 1910.

throw upon the case, the plantar reflex would be normal and there would be no nystagmus or ankle-clonus in hysteria.

The mildness of symptoms in cerebral meningitis may mark it as a case of serous meningitis, but still there may be much headache, vomiting, optic neuritis, convulsions, and coma. The question can only be satisfactorily settled by an examination of the cerebro-spinal fluid. It must, however, be noted that if such an examination often affords valuable information, it may on occasion only increase diagnostic difficulties. If, as in a case the writer saw recently, there is obtained on one occasion Koch's bacillus and an excess of lymphocytes, and on another some polymorphonuclears, the meningococcus, and streptococci, not much advance has been made towards a precise and definite diagnosis.

Tubercular and post-basic meningitis agree in having many symptoms in common; they also have many points of distinction. The latter occurs almost exclusively among children under one year old; its onset is more rapid, and initial fever higher; the head is more markedly retracted; the temperature is higher, and there may be erythematous patches; the upper eyelids are retracted, and the patient may seek the light rather than avoid it, and later there is amaurosis rather than optic neuritis; the joints may be swollen, the abdomen may be flaccid or distended, and the cutaneous sensibility is slight.

Adult sporadic or epidemic cerebro-spinal meningitis is marked off from tubercular meningitis by its more rapid onset, more severe headache, higher fever, much

greater head retraction, pains down the centre of the back, joint troubles, specific rashes, the turbid, bloody or purulent cerebro-spinal fluid, in which there is an excessive polymorphonuclear leucocytosis—this point is fairly reliable if Koch's bacillus is absent from the fluid, but it is not absolute—and, most important of all, there is the presence in the fluid of the meningococcus or pneumococcus, or both.

The general disease with which tubercular meningitis is likely to be confounded is typhoid fever. They may both begin with a general out-of-sorts state, severe frontal headache, pyrexia, and a tendency to drowsiness, and the tâche cérébrale may be as easily obtained in the one case as the other, and, as already mentioned, acute meningitis may develop during an attack of fever. The difficulty of differentiation is most likely to arise in the early days of either disease: the severe headache often present in typhoid fever is particularly disturbing to its diagnosis, but a person suffering from tubercular meningitis is more irritable when disturbed and more restless than a typhoid fever patient; later the contracted abdomen, with constipation in the one case, and the distended abdomen, with its red spots and diarrhœa, in the other, speak volumes. Widal's reaction is effective only when the march of events will have removed difficulties.

Sporadic and epidemic cerebro-spinal meningitis are distinguished from acute purulent cerebro-spinal lepto-meningitis by the more marked head retraction, joint troubles, and rashes of the former, and the presence in the cerebro-spinal fluid of the meningococcus; but the

herpetic eruption on the lips, side of nose, and below the eyes, may be seen in the latter as in the former diseases. It may be impossible by symptoms only to distinguish influenzal from epidemic cerebro-spinal meningitis, unless influenza is rife at the time; intense headache, high fever, and extreme head retraction, are met with in both diseases. Again the presence of the meningococcus would be conclusive.

Infantile post-basic and epidemic cerebro-spinal meningitis have in common head retraction, opisthotonos, progressive emaciation, sudden onset, etc., but they differ in the former being a disease of early infancy and having no herpes labialis or rash; retraction of the upper eyelids is frequent, and optic neuritis is very rare. In the latter optic neuritis is common, and the disease attacks persons of all ages, and herpes and rashes are present with few exceptions.

In syphilitic meningitis, after the clinical symptoms or the finding of the *Spirochæta pallida* in the blood has assured us that the patient is syphilitic, the meningeal inflammation may yet be tuberculous, as we know that syphilitics are prone to become tuberculous; and we should still, therefore, search for Koch's bacillus, as syphilitic meningitis is mostly of a chronic type, in which the symptoms may approach those of tumour of the brain. Those suggestions we made referring symptoms to tuberculous meningitis or intracranial abscess may be helpful here.

Mr. Balance says that when the following group of symptoms are found in conjunction, septic sinus thrombosis is certainly present:

1. A history of a purulent aural discharge for one year or more.

2. Sudden onset of illness, with headache, vomiting, and pain in the affected side.

3. Temperature of the pyæmic type, with rigors, or rigors take the place of vomiting, especially in children.

4. Vomiting frequent, and repeated day by day.

5. Local tenderness and œdema over the mastoid or in the course of the jugular vein.

6. Stiffness of the muscles of the back or side of neck.

7. Optic neuritis.

8. Diarrhœa.

Sir William Macewen says that 'symptoms of disintegrating sinus thrombosis are distinctive of the affection. High temperature, with marked remissions, rapid and weak pulse, repeated and increasingly recurrent rigors, followed by profuse perspiration, symptoms of colliquative diarrhœa, pain in the upper third of the posterior triangle, submastoid region, and down the course of the affected jugular, are often marked'; and 'when infective sinus thrombosis, intracranial abscess, and basal suppurative leptomeningitis all occur together, the symptoms of the first overshadows the others, and is the most important, and first requires attention because of the great probability of infective particles being carried into the general circulation and deposited in the small vessels of the lungs, producing first pulmonary infarction and shortly afterwards abscess, but the last-mentioned condition may even then be recognized by the retraction of the head and neck, and the affection of the intracranial nerves.'

The same syndrome, or general symptom-complex, may be present in most acute intracranial diseases; and it may only be by very careful observation or late in an illness that the doctor notices some symptom or condition which his previous experience and knowledge assure him can, or are very likely to, occur when a specific part of the cranial contents is diseased. Again, instead of having, so to speak, a surfeit of symptoms, he may be misled by their paucity, or, having been accustomed to see certain symptoms in association when a particular disease has been present, he may be led very wide of the mark by their dissociation.

Before we enter upon the consideration of the somewhat complex question of differentiating abscess in one part of the brain from that in another by the phenomena commonly presented by lesions in such parts, it may be advantageous to reflect upon the great similarity of symptoms which other acute diseases of the brain exhibit, in order that we may not too hastily diagnose abscess, and later find, to our bitter regret, that our diagnosis has been erroneous—*e.g.*, deafness, vertigo, nystagmus, high fever, and severe headache, may only indicate disease of the labyrinth.

When brain troubles develop after a long-continued otorrhœa, we can eliminate meningitis, except that which we have termed 'acute meningitis,' the turn of events only showing us whether it is of the serous or purulent type. Absence of pain and tenderness and œdema over the mastoid bone will go far in excluding disease of the mastoid; and, similarly, a normal con-

dition of the jugular vein will almost, but not altogether, exclude thrombosis of the lateral sinus.

The doctor should not be too quick to see brain abscess in a person suffering from an aural discharge, when the patient is also known to have acute or chronic nephritis or valvular disease of the heart, as these diseases may produce many of the symptoms of brain abscess without any such disease being present. Of course, malignant endocarditis, as is mentioned elsewhere, may itself send a focus of infection to the brain and cause abscess ; nor does purulent middle-ear disease, even when associated with headache, vomiting, giddiness, and optic neuritis, necessarily mean abscess of the brain. Barr and Rowan found optic neuritis present eleven times in 160 cases, or 6·8 per cent., which after-events proved were not symptomatic of abscess.

We have endeavoured in the foregoing pages to describe the symptoms of intracranial abscess, cerebral sinus thrombosis, and the various forms of cerebral meningitis, and in the last few pages have made suggestions how the latter are to be distinguished from one another. It now becomes our duty to state how the symptoms of meningitis agree with or differ from abscess or sinus thrombosis, bearing in mind that meningitis, in the form of purulent leptomeningitis, may coexist with either or both of the other diseases.

Septic meningitis so closely resembles intracranial abscess in the matter of general symptoms that we cannot attempt to distinguish between them unless localizing signs of the latter develop or we find facts which can be considered conclusive in cyto-diagnosis.

The state of the pulse is of great moment. The general rule is that the pulse is **quick** and **small** in meningitis, phlebitis, and infective sinus thrombosis, contrasting with the **slow** and **full** pulse of abscess. And the temperature is almost equally important. A febrile temperature of 3°, 4°, or 5°, is almost the constant concomitant of meningitis, while a normal or subnormal temperature is just as certainly exhibited in abscess. Moreover, if the temperature is febrile in abscess it runs a very irregular course, running up in a few hours from normal or subnormal to 105° F., or higher, and in a day, or even sooner, it may sink to its former level; but this varying conduct of the temperature is also significant of epidemic meningitis.

Headache in cerebral meningitis is for the most part general, and, if localized, it is complained of mostly across the forehead, occasionally at the vertex, and perhaps least of all at the occiput, except in epidemic cerebro-spinal meningitis. In abscess of the brain headache is very similar, both in character and situation, to meningeal headache, except in the cerebellar variety, when it has a predilection for the occiput; and when in such cases the pain in the head is continued down the back, and there are also girdle pains and rigidity of both legs, purulent spinal leptomeningitis is also present. Retraction of the head and rigidity of the cervical muscles are seen in their most extreme form in cerebro-spinal meningitis, less so in cerebellar abscess, and they are occasionally present in a mild form in cerebral abscess when there is no meningitis.

Hyperæsthesia of the skin is a distinctly meningeal

symptom, and seen best in tubercular meningitis; but there is nothing in the vomiting, either in the matter of frequency or the time of day in which it occurs, which enables us to say that it is meningeal only, and the same may be said of optic neuritis and the state of the pupils. Retracted abdomen and the tâche cérébrale are almost constant symptoms of meningitis, but they are also occasionally noticed in abscess. Paralyses of the third, sixth, and seventh cranial nerves are often seen together in meningitis, but they also occur together in abscess; but paralysis of the sixth nerve only, when present with other cerebral symptoms, is very suggestive of meningitis, while paralyses of the ninth, tenth, eleventh, and twelfth nerves are not seen in meningitis, but may be observed in extracerebellar abscess. Monoplegia or hemiplegia are seen in meningitis, but only when the patients are far through, while in abscess they may be seen at an earlier stage.

Definite information may be obtained from the examination of the cerebro-spinal fluid, in the scanty or excessive leucocytosis it may present, a count of less than 14,000 per millimetre pointing to abscess and over 17,000 to meningitis, and in the presence or absence of the distinctive micro-organisms.

In the earlier stages of meningitis there is a greater degree of irritability, and the mind is clearer than in the same stage of abscess. The breathing is quick in meningitis, except in suppurative meningitis of the cerebellar fossa, and slow in abscess. Kernig's sign is most valuable as indicative of meningitis, but its value is a little discounted by the fact that it may be produced by

any sort of meningeal irritation, such as hæmorrhage into the subarachnoid space, extradural or subcortical abscess, sinus thrombosis, Pott's disease of the spine, and even rheumatism of the spinal muscles. We have this much assistance in an examination of the urine—that a diminution of the chlorides means inflammatory exudation, and that a urine of high colour and high specific gravity, and containing a great excess of earthy phosphates, indicates destruction of nervous tissue.

As was pointed out in the Introductory Note and under the heading of each disease, the nature of any immediately preceding or concomitant illness points with some exactness to the form of the cerebral trouble.

When we find such a condition as aphasia developed during the course of a disease, other symptoms of which point to the brain or its meninges as the part affected, it might be thought that we have at length arrived at something material, tangible, and absolutely definite, as suggesting a gross cerebral lesion, and a something that must be conspicuous by its absence in meningitis; but this is not the case.

Time and again persons who have suffered from tubercular, acute, or epidemic cerebro-spinal meningitis have become aphasic, and nothing more has been found at the necropsy than a thick exudate over Broca's convolution. Moreover, we find aphasia occurring when there is no lesion whatever, where after-events prove the case to have been functional, or toxic, as in uræmia.

There is some little enlightenment upon the matter in the permanency or non-permanency of the aphasia. If the condition disappears from time to time and again

reappears, it is suggestive of meningitis, as we know that intracranial pressure rises and falls from time to time; or in any case we can conclude that the function, but not the integrity, of the motor speech-centres in Broca's convolution, or, in case of agraphia, the communal fibres in the island of Reil, have not been destroyed.

But the presence of aphasia, or any condition which points to lesion of any definite area or part of the brain, is overwhelmingly in favour of abscess as opposed to meningitis; and when localizing symptoms are exhibited in meningitis they should not be allowed to have any serious counterbalancing power. Their presence, indeed, only suggests that the part to which they refer us is injuriously affected by the results of the meningeal inflammation, or has actually become diseased through contact with the inflamed meninges.

The author has under observation a young woman who has suffered for three years from headache—sometimes over the left half of the forehead, at other times over the right side of the occiput—a gradually increasing loss of sight, and general convulsions. The reflexes are normal, and there is a slow nystagmus. The only localizing symptom that is apparent is the starting-point of the convulsions, which is always in the left hand. The value of the site of the headache is insignificant as compared with this left-handed convulsion, which directs us plainly, whatever the nature of the trouble may be, to the hand-centre in the motor area of the right cerebral cortex.

In any attempt to differentiate between subacute or

chronic abscess and a quickly-growing glioma or soft sarcoma of the brain, the difficulty may be almost too great for us; but it will certainly be to our advantage to remember that in tumours the symptoms do not appear so rapidly one after the other as in abscess, that the optic neuritis is longer in making its appearance and becomes more intense than in abscess, that the usual causes of abscess are wanting, and that such a consequence of abscess as rigors is also not observed.

If we can assume that an illness is due to intracranial abscess, our next thought must necessarily be, Is the abscess without or within the brain? If the pus is extracerebral or extracerebellar, and so circumscribed as to form an abscess, it will be liable to produce just those symptoms which an abscess of the subjacent brain would produce. If the pus is not circumscribed, there will be a more or less diffuse pachymeningitis (dura) with extradural pus, or a purulent leptomeningitis (pia and arachnoid), as the case may be; but the site of the abscess in relation to the different meninges we may be quite unable to decide upon, and if the pus is outside the periosteum, above or behind the ear, we may still have the **general** symptoms of intracranial abscess.

When an abscess is extracerebral, whether extra- or subdural, or the latter be inter- or intrameningeal, if it be otitic in origin it will be found either on the roof of the tympanum, in relation with the temporal lobe, or in the groove of the lateral sinus, in relation with the cerebellum, and in either case be consequent on a chronic otorrhœa. In addition to the constitutional

suggestions, of which a single rigor is an important item, we have also as guides of some reliance headache referred to the site of the lesion, tenderness or pain on pressure and percussion more localized than it is likely to be in intracerebral, circumscribed œdema or tumefaction, with possibly such localizing symptoms as spastic contracture or paresis of muscles. We also know that such abscesses are likely to be caused by violence, usually penetrating wounds or local diseases of the skull; they are acute, and run a rapid course. Intracerebral abscesses are more likely to be caused by disease of the middle ear and nasal cavities, and by indirect infection.

We have now to decide between the cerebrum and cerebellum as sites of abscess, and it may be that the symptoms will be so characteristic of the one or the other that even those who have paid no particular attention to this class of disease will have little difficulty in saying to which variety any particular case may belong; while in other cases those who have made acute diseases of the brain a special study can make no conclusive diagnosis upon the evidence of the symptoms exhibited, being able to go no farther than to say it is abscess of the brain.

Some symptoms are equally common to both sites; others point to one rather than the other, while a very few are absolutely peculiar to one or other site.

The syndrome, or group of symptoms, common to cerebral and cerebellar abscesses are: Headache, when general or confined to the forehead; vomiting, with a furred or glazed tongue; dulness; restlessness; irritability;

stupidity, slow cerebration and drowsiness, passing into coma; optic neuritis; deafness; slow pulse and sub-normal temperature (which, however, may not be present in either variety); slow, laboured, or sighing breathing; normal or exaggerated knee-jerks. The pupils are sluggish and dilated in both cases.

Symptoms favourable to One or Other Site.

A dorsal decubitus points to the cerebrum, and a curled-up position upon one side to the cerebellum. Convulsions occur all through in cerebral disease, and may be unilateral, late in cerebellar disease, and have no characteristic *locale*.

Paralysis of the extremities, when spastic, points to the cerebrum; when flaccid, it is in favour of the cerebellum. Vertigo is rarely seen in cerebral abscess, but is frequent and very intense in many cases of cerebellar abscess. Sudden stoppage of the respiration while the heart still beats is peculiarly characteristic of cerebellar disease, but not absolutely so. Hemianopsia is very uncommon in cerebellar abscess, and only occurs when the abscess is of large size and presses upon the occipital lobe. Inco-ordination of the upper extremity when the patient is in bed is highly suggestive of cerebellar disease, but is also possible in abscess of the frontal lobe. Mr. Balance says that in frontal ataxia a man sways to the right or left when he stands without assistance, but he does not describe zigzags or stagger in his walk, like a person with a cerebellar lesion. The younger the patient, the greater is the probability of the abscess being cerebellar. The absence of localizing

signs may compel the surgeon to depend on the direction of the extension of the disease while he is removing necrosed temporal bone. Ankle-clonus and an extensor plantar reflex are more likely to be cerebral conditions.

If we can assume that an abscess is cerebral, we have still to determine, if we can, (1) the **hemisphere** in which it is located, and (2) its **locale** in that hemisphere.

1. Symptoms which point to the Hemisphere affected.

Pain localized to one particular spot and increased by pressure suggests that part as the site of the abscess, and that the latter is near the cortex, if it is not, indeed, extracerebral. The earlier or greater degree of optic neuritis upon one side is slightly suggestive of that side being the diseased one, as also is a divergent squint, or a more dilated pupil, or the cicatrix of an old injury or indentation of bone. Hemiplegia and monoplegia, whether the latter be brachial or sural, point to the contralateral side. Aphasia and other speech defects point to the left hemisphere as the diseased one, except in left-handed people.

2. The Locale in the Cerebral Hemisphere.

Temporal or Temporo-Sphenoidal Lobe Symptoms.—The temporal lobe is limited behind by an imaginary line drawn from the parieto-occipital fissure to the pre-occipital notch, in front by the Sylvian fissure, and above by an imaginary prolongation of the horizontal limb of the fissure of Sylvius to the first imaginary line. It will be considered first, as it is the site of abscess far

more often than any other part of the cerebrum. The most reliable focal symptom is **sensory** aphasia, but it is not always present, as the greater part of the lobe is not concerned with speech.

In **sensory** aphasia, in which the speech defect is on the perceptive side, the patient does not comprehend the meaning of spoken words (auditory aphasia, or word-deafness), although he may be a great talker, nor sometimes of written words (visual aphasia, or word-blindness); it is also often associated with some degree of motor aphasia, the interference with the perception of spoken or written words injuriously influencing the motor or expressive power. It is not usually accompanied by hemiplegia, yet there may be paralysis, and whatever form it takes it is on the contralateral side, and it results from pressure transmitted from the temporal lobe to the motor centres of the ascending frontal convolution from below upwards.

When this form of aphasia is present, the abscess is in the posterior half of the first (or superior) left temporal convolution.

If the aphasia is that of recollection, in which the patient is unable to name correctly objects shown to him, the same convolution is in fault. When the abscess approaches the internal capsule, the sense of touch is deadened on the contralateral side.

Paralysis of the sixth nerve is very likely to be present, as well as complete oculo-motor paralysis, both being homolateral.

There may be hemianopia from extension of the suppuration into the white matter. Nystagmus is

uncommon; if it occurs, the abscess is pressing upon the parietal lobe.

The sense of smell, the centres for which are situated in the uncinate gyrus of the same side and to a small extent of the opposite side, may be diminished, as in failing to distinguish the scent of roses from that of violets, or lost, as in failing to perceive the smell of garlic, valerian or asafœtida.

In connection with deep-seated lesions of this lobe, mention must be made of the dreamy state, in which the patient is drowsy and apparently unconscious of his surroundings, and stares into vacancy.

The centre for hearing is situated in the middle of the superior temporal convolution on the side opposite to the deafness which injury to it causes; therefore, in abscess of the temporal lobe the patient may be deaf in both ears, but from different causes—on the homolateral side from disease in the temporal bone, and on the contralateral side from destruction or injury of the cerebral centre for hearing. Abscess of the anterior part of the lobe is very likely to cause voracious hunger and thirst, conditions which have been recorded in a series of seventeen cases of traumatic abscess located here; but as they have been observed in lesions of the prefrontal lobe as well as of the cerebellum, they are not reliable as focal symptoms.

Frontal Lobe Symptoms.—As the frontal lobe is of large size, extending on its outer surface from the anterior pole of the brain to the fissure of Rolando, few surgeons would be desirous of operating upon it in case of abscess, unless they had something definite to guide

them as to which part of the lobe was affected. Fortunately, such guidance is possible.

The signs characteristic of prefrontal lobe lesions, abscess included, are: *First*, deterioration of the mental character, from mere inability to give attention, loss of memory, or a tendency to joking or loquacity, to serious mental breakdown; and *second*, incontinence of urine. Another characteristic, less important probably, but still one having a distinct value, is a stumbling, not ataxic, gait, attributed to a paresis of the muscles of the lower sacral region (rump).

The fact that abscesses in this lobe are well known to follow infective injuries of the forehead must be kept in mind, but not allowed a significance over symptoms which point elsewhere—*e.g.*, sensory aphasia.

A frontal lobe abscess is very likely to be a chronic one, and may therefore be surrounded by a thick capsule; and in lesions here optic neuritis may be late in its oncoming, with the exception immediately to be stated, and be more intense on the side of the lesion.

If abscess starts on the basal or orbital plate of the frontal bone, optic neuritis may be early, the sense of smell will be interfered with or destroyed, and the importance of this point is greater if the loss be one-sided. The diagnosis of abscess here would be more positive if disease of the ethmoidal cells could be proved. The anterior third of this lobe is the part of the brain most likely to be affected by frontal or fronto-maxillary sinus abscess.

The psychical troubles already spoken of are more frequent in lesions of the **left** prefrontal lobe.

Motor aphasia, in which the speech difficulty is in expression, the patient being speechless, or almost so, unable to talk to himself or read to himself, knowing the words he wishes to speak, but unable to articulate them—that is, spontaneous speech and also the capacity to repeat are lost—points to the lesion being in the posterior part of the third left frontal convolution, and in the adjacent part of the ascending frontal, or the conducting paths from the cortex (communal paths from the speech-centres), always bearing in mind, whatever form of aphasia be present, that if the patient be left-handed, the right hemisphere will be the part affected.

This form of aphasia is often associated with hemiplegia, but with or without hemiplegia it constitutes a most reliable localizing symptom. It must, however, be **constant**, and it may not be noticed after the second stage of abscess has been entered upon if mental dulness be marked; if the aphasia be transient, it points to the parts between the speech-centres and the internal capsule.

Although we know that the motor speech-centres are in the vast majority of cases located in Broca's convolution—that portion of the cerebrum which surrounds the vertical and horizontal divisions of the fissure of Sylvius, the **pars triangularis**—aphasia must not be looked upon as absolutely necessary in lesion there, several cases being on record in which the patient could talk with perfect fluency and correctness, and yet the area which Broca designated as the site of the speech-centres was destroyed.[1]

[1] *Vide British Medical Journal*, March 15, 1879.

Motor agraphia, in which a person cannot with either hand write to dictation or copy print into writing, indicates trouble in the posterior end of the second frontal convolution. Agraphia is always associated with aphasia, and when we reflect how near to each other are the centres for the two, it is no matter of surprise; but why aphasia should be present—and many cases have been recorded and the diagnosis verified after death—without agraphia is not obvious.

In abscess of the post-frontal or posterior third of the frontal lobe there would be the paralyses which are consequent on the implication of the motor centres in the ascending frontal convolution—viz., from above downwards, leg, trunk, arm, hand, face, mouth.

In lesions of the **white substance** (intramedullary portion) of the frontal lobe, there may be fine rapid tremors of the fingers, worse on the side of the lesion; general convulsions, with loss of consciousness, the fits starting in fingers, arms, or legs; hemiplegia and weakness of face, with a tendency to fall on the same side (Beevor on 'Tumours of the Brain').

Parietal Lobe Symptoms.—The parietal lobe includes that portion of the brain between the fissure of Rolando in front, the parieto-occipital fissure behind, and the horizontal limb of the fissure of Sylvius below.

Apraxia, in which the patient cannot make the correct movements for the action he desires to perform, although he may neither be paralyzed nor ataxic, points to lesion in either parietal lobe. Hemianopia may be present. The lessening of pain and heat sensibility,

analgesia, and thermo-anæsthesia, are contralateral to the lesion.

Astereognosis, the inability to recognize objects by handling them while the eyes are closed, indicates a lesion in the superior parietal convolution, and particularly its anterior portion, and the limb affected is contralateral to the cerebral lesion.

Sensory agraphia (often incomplete), in which a person cannot write words because he cannot call them to mind, depends upon a lesion in the **left** angular gyrus or supramarginal gyrus.

Alexia, the inability to read, and **amusia**, the inability to produce or appreciate music, both point to the left parietal lobe.

Dyslexia, the inability to read aloud or to oneself more than a few words consecutively, points to a lesion in the left parietal lobe, in or near the angular gyrus.

Occipital Lobe Symptoms.—This lobe includes that part of the cerebral hemisphere behind the lateral portion of the parieto-occipital fissure and a line connecting the extremity of this fissure with the pre-occipital notch, and abscess in it is usually connected with pyæmia; but it may result from violence, and has been known to ensue after disease of the middle ear.

Eyesight depends upon visual centres in the cortex of this lobe, disease in which produces focal symptoms concerned with the abnormality or loss of the sense. These centres are of two kinds, and are named 'primary' and 'higher' or 'secondary.' The **primary**, which enable a person to see, are situated in the cortex of the

mesial surface of the lobe—the cuneus, a wedge-shaped area between the calcarine and internal parieto-occipital fissures—and their destruction causes blindness, which may be a shaded darkness or an absolute blackness, limited to about half the field, and therefore called 'hemianopia,' in which a person only sees half the object he is looking at, half the page he is attempting to read, and so on, and the result is the same whether he looks with both eyes or only one; and as the half-blindness is on the same side in each eye—the temporal side in the one eye and the nasal in the other—it is called **homonymous**. In **nasal** hemianopia the inner, and in **temporal** hemianopia the outer half of the visual field is deficient. If the lesion is small a quadrantic or sectional defect only ensues, but it is still termed 'hemianopia,' if the deficiency occupies a symmetrical position in the visual field of the two eyes. The **higher** visual centres, which enable a person to recognize, are located in the posterior end of the inferior occipital convolution and adjacent parts of the parietal and temporal lobes, and lesions of them cause mind-blindness, or an inability to recognize familiar objects and persons. Both varieties may be diseased at the same time, when there would be homonymous hemianopia plus mind-blindness, and lesions of the primary centres in both lobes would cause total blindness.

Dr. Wollaston, in the fourth decade of last century, drew renewed attention, after Newton, to half-blindness in a paper on 'Semi-Decussation of the Optic Nerves,' published in the Philosophical Transactions of that

time. It is well known to be an occasional feature of migraine, and disappearing with it. Dr. Wollaston himself suffered from the trouble, an attack lasting from about fifteen to twenty minutes, and then was removed suddenly and entirely; and one of his medical friends had also been subject to transitory attacks of the same nature for seventeen years, whenever his liver was out of order. It may be that these transitory attacks of half-blindness do not always owe their origin to trifling disorders, but may sometimes depend upon organic disease of some part of the nervous structures, which transmit nervous stimuli from the retina to the cerebral cortex and convey visual impressions back from the brain to the retina. In this doctor's own case, although he suffered for years only from transitory half-blindness—whether it was permanent for some time before death Dr. Mackenzie, who relates the fact in his work on 'Diseases of the Eye,' does not record—the right optic thalamus, corpus striatum, and optic nerve were extensively diseased. In transitory temporal hemianopia the cause is a passing acute intracranial pressure.

It is necessary to be able to distinguish the hemianopia due to lesions of the primary visual centres from that consequent to a lesion of the optic nerves, optic tract, optic thalami, optic radiations, the internal capsule and the basal ganglia.

For this differentiation, we partly rely upon the pupillary reflex, first pointed out by Wernicke—if the pupil contracts actively to light on the blind side of the field, the lesion cannot be in the tract; and if the

pupil does not so react, the lesion must be in the tract—that is, the break in the conduction must occur where the fibres to the oculo-motor nucleus are given off, and the nervous structures beyond this point will be normal so far as hemianopia is concerned.

If the visual defect occurs only in one eye, or unsymmetrically in the two eyes, the lesion is in front of the optic commissure—*i.e.*, in the optic nerve of that side after semi-decussation has occurred. If the hemianopia is temporal on both sides, the lesion is in the chiasm. If the hemianopia is homonymous, but only sectionally so, the trouble is beyond the chiasm.

If we have been able to rule out the optic tract, we may conclude that hemianopia probably depends upon an occipital lesion, if it is unaccompanied by hemiplegia, motor aphasia, or paralysis of the cerebral nerves.

The foregoing remarks only refer to visual defects which do not offer ophthalmoscopic evidence of disease of the deep tissues of the eye.

Other forms of curtailment of the visual field occur, the **concentric**, and **scotomata**, whether central or isolated, are due to diseases or impaired function of the eye itself, and not to intracranial disease, and therefore do not concern us.

The colour sense is in the posterior part of the superior and inferior occipito-temporal convolutions.

Colour-blindness may be congenital, and may be due to hysteria, but in any case it is not a focal symptom of reliability; we cannot differentiate the colour-blindness due to one part of the central ocular system from that of another, as we can do with hemianopia.

If the **aura** which often precedes a convulsion consists of visual spectra or flashes of light, possibly of different colours, the fact at once refers us to the occipital lobe. The spectra may be seen by both eyes or only by one, and they may be hemianopic in character.

Symptoms Directly Suggestive of the Cerebellum.

Lateral nystagmus, said to be always present at some stage of abscess, is perhaps the most valuable of cerebellar symptoms, and if not present may often be elicited by turning the patient's head towards the side of the lesion. Given only this one localizing symptom, with concomitant general ones of intracranial abscess, its presence is clearly in favour of the cerebellum as the site. Other and scarcely less important symptoms are vertigo associated with vomiting, occipital headache, frequent opening and closing of the mouth, widely dilated pupils, and a highly resonant percussion note obtained by tapping over the pterion, or just behind it, and denoting distended lateral ventricles, when they occur together or can be elicited. In no way less suggestive are excessively low temperature and slow breathing, difficult and jerky articulation, difficult deglutition, paralysis of palate or tongue, all indicating pressure upon, or involvement of, the bulbar centres; rigidity of nuchal muscles and susceptibility to pressure on the neck; retraction of the head, with the face pointing to one shoulder, and the occiput resting upon the other; conjugate deviation of the eyes to one side; tendency to fall to one or other side, and **hemiataxia** and **hemiparesis**; but the latter may only be shown by

the muscles of one side being less firm than on the other, in the arm less grasping power, in the leg less power to stand, than in those of the opposite side.

If the *locale* of an abscess is cerebellar, in which hemisphere is it, or is it in the vermis? Optic neuritis more intense upon one side, or beginning earlier upon one side, is in favour of that side as the side of the lesion; if the pupils are unequally dilated, with rare exceptions the abscess is on the side of the greater dilatation. Tremor and convulsive movements may be on either side. The patient is likely to be curled up in bed on the affected side, but the rule is not absolute.

Symptoms Directly Homolateral.

The affected side is indicated by any of the following conditions:

The direction of the more rapid oscillations in horizontal nystagmus; hemiataxia or inco-ordination of movements in one hand or foot; localized pain and tenderness on pressure; hemiparesis, or weakness of the muscles of the limbs of one side, especially of the arms; cranial nerve paralysis, other than the third and seventh, which may point to either side; the side of the mastoid abscess, or ear trouble, or greater deafness, unless there has been previous aural disease on that side; tendency to sway or fall to one side; when the occiput is upon one shoulder and the face looks to the opposite one, the side to which the face turns, and tremor or convulsive movements in those parts governed by cranial nerves. In diadokokinesis—meaning successive movements—when the patient attempts rapidly alternating

pronation and supination, as by laying his hand on the table and quickly turning it over and back again, he performs the action slowly or not at all on the side of the lesion.

Contralateral signification is forced rotation in a vertical axis.

Symptoms Directly Suggestive of the Vermis.

The symptoms are equally distributed on both sides: there is a tendency to fall backwards or forwards, and a sensation of falling when sitting up in bed, and vertigo and vomiting are induced by the sitting-up position; head retraction and opisthotonos, likely to be accompanied by seventh and eighth nerve paralysis; incoordination is general. Sudden death is more possible than in abscess of the lateral lobes.

We are now primarily concerned with abscess of the cerebellum, and have incidentally spoken of tumours; but there are other abnormal conditions of the organ in which its characteristic symptoms are exhibited.

Atrophy needs only the merest mention. It is said to be present only in those of impaired intellect or actual imbeciles, and is due to a defect in the development of the organ; the granular layer of the cortex may be absent, or the whole organ be almost non-existent. The symptoms are chiefly manifested in the peculiar walk and hesitating speech.

Hæmorrhage (apoplexy) and thrombosis are possible, if rare, affections, and the softening consequent upon the latter, both in the organ itself and adjacent

part of the medulla oblongata, is of far-reaching importance.

The symptoms of hæmorrhage, usually from the superior cerebellar artery, may easily be looked upon as referring us to basal meningitis. They develop quite suddenly. The patient often utters a loud scream, and at once complains of dizziness, and vomits ; headache, occipital or frontal, is complained of, and head retraction, boat-shaped abdomen, and Kernig's sign are observed; vomiting persists, and he may scream at intervals; there is no loss of consciousness. He will probably lie curled up on the affected side; the pupils will be widely dilated; and he may die within a couple of days or live as many weeks, according to the volume of hæmorrhage, death occurring suddenly or by sudden cessation of the respiration, while the heart for a time continues to beat. The keynote to the condition is the suddenness of onset, the characteristic attitude, the ataxic gait, with the legs wide apart if he is still able to walk, and finally the respiratory failure.

In **thrombosis** it is usually the posterior inferior cerebellar artery which is diseased. The onset is sudden ; vomiting comes on at once and persists; there is much occipital headache, but no loss of consciousness; there will be a tendency to fall to one or other side, and ataxia of the arm and leg of that side, and there may be nystagmus. If the patient lives a few weeks, there will be, probably on account of the consequent softening spreading to the medulla oblongata, crossed dissociate anæsthesia, particularly for pain and heat, of the face (the distribution of the fifth nerve) on

the homolateral, and of the arm, side of trunk, and leg on the contralateral side, with paralysis of the muscles of deglutition and of the soft palate on the affected side and paresis of the limbs on the opposite side.

The disease eventually ends fatally. The symptoms are significantly cerebellar, but are differentiated from those of abscess by the suddenness of onset, the absence of the usual causes of abscess, and the presence of possible causes of thrombosis or embolism, such as debased states of the blood, as obtains in anæmia in young people. Recent syphilis is the most frequent cause of the narrowing and ultimate occlusion of arteries in early manhood and the prime of life, arteriosclerosis in the later years of life, and valvular disease of the heart at any age.

There remains the important question of distinguishing between cerebellar abscess and labyrinthine suppuration, whether they are in association, or only the latter be present.

In labyrinthine suppuration, when the cerebellum is not affected, the headache is not so severe and continuous, and there is not the same degree of dulness, drowsiness, or tendency to coma as there is in abscess; but still there is headache, and also vertigo, vomiting, unsteady gait, Romberg's symptom, perhaps spontaneous horizontal nystagmus directed towards the affected side and made worse on extreme deviation of the eyes to that side; and optic neuritis has been mentioned as being present. The symptoms being so very similar, the direction of the nystagmus is all-important.

In cerebellar disease the nystagmus may be to either side, but is usually towards the side of the lesion, while 'in labyrinthine disease it is first towards the side of the lesion, but as the disease progresses it turns towards the sound side, and after complete destruction of the working power of the labyrinth, nystagmus ceases' (Neumann).

Dr. Barr, senior, of Glasgow, distinguishes the nystagmus of the two diseases as follows: In cerebellar suppuration nystagmus is **slight** at first, and becomes more pronounced as the disease progresses, while labyrinthine disease is **most marked** at first and gradually disappears.

Bardny's 'caloric' or 'vestibular' test may settle the point as between a sound and a diseased labyrinth. If cold and then hot water be poured into the ear, the normal labyrinth will give no sign of irritability—*i.e.*, the existing nystagmus will not be intensified—and labyrinthine disease can be excluded.

It may be well to keep the following facts in mind; the symptoms given are not the only ones possible, but they are probably the most characteristic:

Lesions, of which abscess is a possible one, in the pons Varolii, medulla oblongata, corpus striatum, optic thalamus, and pituitary body, are inoperable; but urgent symptoms, such as headache and optic neuritis, may be relieved by decompressive operations.

Retrobulbar neuritis may be caused by abscess of the posterior ethmoidal cells, although there may be no empyema of those cells. Lesions in the **uncinate**

gyrus have been known to produce a horrible odour in the mouth, or a noxious taste on one side of the tongue, along with dreamy sensations, great dread, faintness, giddiness, and other symptoms of a focal character.

Lagophthalmos (λαγος, a hare; and οφθαλμος, the eye), the act of sleeping with the eyes wide open, as a hare is supposed to do, is absent or very slight in diseases affecting the internal capsule, and markedly present when the portio dura in the pons is affected.

Hemianæsthesia is common to destructive lesions of the medulla oblongata, crura cerebri, the posterior third of the hinder segment of the internal capsule (in disease of the anterior third, if hemianæsthesia is present, it is transitory); and there will be hemianopia if the nerve fibres connected with vision are involved, in unilateral disease of the spinal cord, and in hysteria.

Hysterical hemianæsthesia is sharply defined, may be —and, indeed, is often—absolute, may flit to the opposite side and return again to its original site, and is not usually accompanied by motor paralysis. There is usually hypersensitiveness of the ovaries, and there may be loss or impairment of the special senses of smell and taste, uniocular diplopia, not dependent upon any error of refraction or disease of the ocular fundus; there is no hemianopia, but concentric contraction of the visual field, which tends to increase as the testing proceeds, is very possible; and perhaps there may be loss or inversion of colour vision, first for violet, then blue, and lastly red.

It may be remarked of hemianæsthesia supposed to be due to hysterical or functional causes that it often

depends on organic causes, as in lesions of the optic thalami.

In a case of hysteria in a single woman, twenty-five years of age, seen by myself some years ago, anæsthesia of leg, arm, and trunk muscles of one side was as sharply limited by mid-spine as if the origin had been organic nervous disease. The case was no doubt of some clinical interest, and I regret that my notes of it are meagre. I have no record of amblyopia or interference with colour sight, but the ovaries were excessively sensitive, and the patient had been witness to a violent hysterical outburst in a fellow-servant a short time previously. Recovery ensued in three weeks after removal of the patient from the care of her too sympathetic mistress.

Crossed paralysis is common to lesions of the pons Varolii, medulla oblongata, and crura cerebri; but the cranial nerves involved differ, and in the two last named there may be hemianæsthesia.

In abscess of the **pons Varolii** hemiplegia may be present on the contralateral side, with **seventh-nerve** paralysis on the homolateral; in cortical lesions they are both on the contralateral side, or if the abscess is large, there is bilateral paralysis of face and limbs. In conjugate deviation, when the pons is involved, the eyes in paralysis turn **from** the lesion, and in spasm from disease of the cerebral cortex **towards** it.

In lesions of the **medulla oblongata** there is likely to be unilateral anæsthesia of the face or paralysis of the palate or vocal cord, or the three conditions may be present, on the side of the lesion, coupled with

anæsthesia of the **opposite** trunk and limbs. If the hemianæsthesia occurs without hemiplegia, the lesion is on the dorsal part of the medulla; if with hemiplegia, the ventral surface is also affected.

Crus Cerebri.—Crossed hemiplegia occurs in lesions in this site—*i.e.*, paralysis of the **third** nerve, ptosis, etc.—upon the homolateral side and hemiplegia on the contralateral, but the two paralyses must occur in association.

Optic Thalamus.—Lesions here produce involuntary movements and ataxia of the opposite limbs, plus hemianopia, if the posterior inferior part of the thalamus is affected. There may also be severe pains in the limbs and hyperæsthesia of the skin.

In lesions of the **corpora quadrigemina** there is usually complete bilateral ophthalmoplegia. If such a degree of paralysis is wanting, the upward movements of the eyes are weakened, and of those nerves which when paralyzed cause fixity of the eyeball (the third, fourth, and sixth) and loss of function of the iris (the third and sympathetic), the fourth, which supplies the superior oblique, is most often paralyzed. Sight and hearing will be seriously affected, and nystagmus may be exhibited; the pupils will no longer respond to light, and perhaps not to accommodation. If it is the posterior bodies, the testes, that are diseased, there will, on account of the proximity of the cerebellum, be early ataxia, with a tendency to fall forwards.

APPENDIX A

LUMBAR PUNCTURE AND ITS USES

LUMBAR puncture was first introduced by the German neurologist Quincke in 1884, as a therapeutic agent in the treatment of tubercular meningitis, in order to lower intracranial pressure by the slow withdrawal of cerebro-spinal fluid, and was first practised in England by Dr. Essex Wynter.

Normal cerebro-spinal fluid is clear and transparent; has a specific gravity of 1006 to 1007, and a slightly alkaline reaction; contains no corpuscular elements, except, perhaps, a few lymphocytes, and only a trace of albumin in the form of globulin, known to be so by the fluid giving no reaction for a proteid substance when it is saturated with sulphate of magnesia. Boiling the fluid, or the addition of acetic acid to it, will give a cloud which, if no further test were applicable, would be equally significant of the presence of albumin or a compound proteid, as mucin; 1 or 2 minims of nitric acid will remove the cloud if due to mucin. When true albumin is present, it signifies disease, as in meningitis and the parasyphilitic nervous affections. Fibrinogen is also absent, as there is no heat-coagulum at a temperature of 56° C., and no fibrin is formed on the addition of serum or a fibrin ferment. The fluid also contains urea, increased in uræmia, and a reducing agent, probably dextrose, which is much in-

creased in diabetes, and is characteristically absent in the various forms of meningitis, always being absent in purulent meningitis, present in small quantity in the early stage of tubercular meningitis, but disappearing later. It has been obtained in crystalline form, and called by Michael Foster 'pyrocatechin.' Although this substance reduces salts of copper, as in Fehling's solution, it is not a true sugar, as it does not ferment with yeast, reduce bismuth, or rotate the plane of polarized light.

The solids amount to about 1 per cent., chiefly chloride of sodium. There are also traces of carbonate and phosphate of sodium.

Cerebro-spinal fluid exists normally in the subdural and subarachnoid spaces, under a mercury pressure in the recumbent position of 40 to 60 millimetres, and a water pressure in the child of 20 to 40 millimetres and 120 millimetres in the adult, which is increased in the sitting posture and still more in disease. Over 180 millimetres is abnormal and 400 millimetres is not unusual, and over 700 millimetres has been observed.

Its average volume is very difficult to estimate. Different observers have placed it at 2 and 5 fluid ounces, but it is dependent upon the pressure of blood at the moment in the cerebral sinuses and veins being increased when the volume of the brain is diminished. Hence its greater pressure in meningeal inflammations.

How this fluid is generated and what becomes of it, as we know it is formed continuously and in considerable quantities, are points which have much exercised the minds of those who have studied the subject; but the absence from it of true albumin and fibrinogen destroy the theory that it is simply an exudation from the blood. Some physiologists favour a transudation from the choroid plexuses of fine capillaries which line the lateral ventricles of the brain. A more correct conclusion is that it is a true secretion from the columnar epithelial cells of the

ependyma which covers the choroid plexuses of the lateral ventricles, from which it passes through the foramen of Magendie to the spinal subarachnoid space at each cardiac systole, and its volume is further increased by the ciliated epithelial cells which line the ependyma of the central spinal canal. As the space in which the fluid occurs is limited, and, as we will show presently, the process of its formation is increasing, it must re-enter the venous circulation. Doubtless the agency of the Pacchionian glands, which form a route to the cerebral veins and longitudinal sinus, plays an important, but still only a supplementary, part, as these bodies do not exist in animals and young children. Cushing and Leonard Hill believe that the fluid finds an exit from the walls of the longitudinal sinus. Others hold that it passes from the subarachnoid space by the lymphatic vessels of the cranial and spinal nerve roots, into the lymphatics of the nerves in their course, and finally, by the lymphatic system, into the thoracic duct and venous circulation.

Opportunities for forming an opinion of the normal fluid are obtained on the first tapping of a spina-bifida, and the questions of its continuous development and the quantity produced daily may be studied in cases of fracture of the skull, when there is also rupture of the tympanic membrane, and in cerebro-spinal rhinorrhœa, in which, after a person has suffered a long time from headache, vomiting, drowsiness, and other meningeal symptoms, there is complete relief on the establishing of a constant dripping from the nose of a clear fluid, which responds to the tests for cerebro-spinal fluid. In the case of a young girl[1] twelve to thirteen years of age the fluid dripped at the rate of three to six drops per minute, or from 12 to 16 ounces per day.

[1] *Vide* case recorded by Dr. Norman Pike, *British Medical Journal*, May 7, 1910.

The chief objective or function of the fluid is to fill up the crevices and spaces of the bony structure in which the central nervous system is situated, placing a fluid medium between the bone and nervous structures, lessening or doing away with the jolting or violence which would otherwise be inevitable.

Choline (from χολή, bile), a ptomaine ($C_2H_{15}NO_2$), is a natural constituent of brain tissue, but an abnormal one of cerebro-spinal fluid, and its presence indicates nerve degeneration. It has been found in cases of cerebral hæmorrhage, in the majority of cases of tabes dorsalis and Jacksonian epilepsy, but it is not found in hysteria or where there is no sign of nerve degeneration. Choline and phosphoric acid may be present in diseases due to chronic alcoholism, both being products of the disintegration of lecithin, which exists in the myelin of medullated nerve tissue. The detection of choline may distinguish between functional and organic nervous diseases, but the test is far too elaborate for any but physiological experts. Mr. Webster says: 'There is little hope of detecting by the micro-chemical tests in use the very minute quantities—fractions of a milligramme—of choline that might be set free from degenerative nervous tissue, and so get into the circulating blood of man or animals in disease of, or after operations upon, the nervous system.'[1] Kaufman isolated no choline from a litre (35·39 fluid ounces) of cerebro-spinal fluid, collected from various patients with nervous diseases.

An anæsthetic is unnecessary for the performance of lumbar puncture except in very young children and excitable persons. The patient is placed in such a position as will put the spinal column on the stretch—either on his hands and knees, or on his left side, with head and shoulders bent and knees drawn up, or sitting astride a chair and

[1] Epitome No. 70, *British Medical Journal*, vol. i., 1909.

leaning over the back; but the last position is least desirable, and, indeed, is fraught with danger, as sudden syncope is very possible, and should only be chosen when dyspnœa or obesity compels. Of course, a child must not see what is going on, and a towel lightly held over the face, with a nurse raising the upper end and speaking words of comfort, has the effect of keeping him quiet.

Carry out the strictest antisepsis with regard to the skin over the spine; make the skin aseptic and the point of insertion of the needle insensitive with a drop of carbolic acid, and place patient on a sterile towel. A hollow needle, or trocar and cannula, made of plain steel, or steel tipped with platinum, and previously sterilized, and from 2 to 3 inches in length, according to the age of the patient, and having the necessary fitting for the accurate adjustment of a syringe in case injection is to follow, is then passed from 1·5 to 3 centimetres ($\frac{2}{5}$ to $\frac{4}{5}$ inch) into the third or fourth lumbar interspace, which is just above or below a line drawn by placing a towel over the lower part of the back from the highest part of one iliac crest to another, at a point one finger's breadth from the middle line, and sharply upwards and inwards towards the middle line of the spinal column, thus obviating as far as possible the chance of causing hæmorrhage or of wounding the cauda equina—or, in case of spinal anæsthesia being required, only obtaining a one-sided anæsthesia. The point of the needle will receive a slight check both at the ligamentum subflavum and the dura mater, and the latter may be pushed forward without being pierced if the needle has not been pushed sharply home.

The cannula having been withdrawn, the puncture is closed with sterilized gauze and collodion—that is, if cytodiagnosis or therapeusis has been the objective; and the receiving vessel for the former and the test-tube and water that may be necessary for the making of a solution for the latter must be previously sterilized. If anæsthesia has

been the objective, as the child is already on a sterile towel, the puncture can be dealt with after the operation for which it has been required is finished.

For analysis 3 c.c. to 5 c.c. (30·4 to 50·7 minims) will be sufficient, but for the relief of intracranial pressure 15 c.c. to 20 c.c. will be required; and if headache or vomiting begin, the needle should be at once withdrawn.

If the point of the needle is obstructed by bone, withdraw and reinsert it, or finally reintroduce in another intervertebral space. If still no fluid is obtained, it is either because the needle is carrying the dura mater forward, the pus is too thick to flow, or there is none present in the spinal subarachnoid space on account of the closure of the foramen of Magendie. If, on introduction of the needle, the patient suddenly screams out or complains of pain, you will have injured a nerve root, and the needle must be withdrawn and reinserted.

It is good practice to reject the first c.c. of fluid, as it may be a little bloody; but if blood flows freely, you have either injured a vessel or the fluid is actually hæmorrhagic. 'After being centrifugalized, the fluid takes on a yellowish colour if it is hæmorrhagic, and retains its normal appearance if the hæmorrhage is accidental' (Mathieu).

The fluid in disease may be clear, turbid, purulent, so thick that it will not run, or it may be bloody. It may pass into the receiver (which must have been sterilized if the fluid is required for examination) in a uniform flow, as in health, or irregularly and in spurts, which will be indicative of a high degree of pressure, as in many cases of cerebral meningitis and sinus thrombosis. It should be allowed to escape slowly, and when it is too thick to flow normal saline may be injected, and repeated if necessary, in order to lessen the consistency.

Lumbar puncture is now much used for diagnosis, and in **cyto-diagnosis**—a name given by Vidal and Ravaut 'to determine the character of a morbid fluid from the study

of its cellular elements'—we have obtained a new field for research that has already been of the highest service. Other uses are the injection into the spinal theca of medicinal agents for the production of a limited anæsthesia and for therapeutic purposes.

Cyto-Diagnosis.—Cerebro-spinal fluid is examined to ascertain the number of leucocytes, and whether they are polymorphonuclears or mononuclears, and for the presence or absence of micro-organisms, for which purposes, before the microscope can be brought into use, the fluid must be centrifuged for at least ten minutes in a test-tube attached to an instrument that revolves 3,000 times per minute, the deposit only being examined. 9,000 leucocytes per milligramme is normal, while 15,000 to 20,000, or more, constitute leucocytosis.

The normal number of lymphocytes or lymph corpuscles are two or three to the field after centrifugalization of the deposit, and under a magnifying power of 400 diameters; a greater number suggests tubercle or syphilis, and their absence is suggestive of functional neurosis, psychosis, and the various forms of neuritis.

Spinal Anæsthesia.—A further development of the uses to which lumbar puncture lends itself was worked out in the matter of anæsthesia, the idea being that if analgesics were introduced into the spinal subarachnoid space the resulting anæsthesia would perhaps be safer than that produced by inhalants, in that it might not produce such grave circulatory and respiratory disturbances. Dr. Coring, I believe, was the first to put the idea into practice; but it did not receive much favour until Dr. Bier, in 1898, revived the practice. Cocaine, in a 2 per cent. solution, was the first analgesic to be applied, and this proving to be wanting in safety, other drugs of a similar kind have had a trial, such as pure stovaine, tropo-cocaine, novo-cocaine, and adrenalin with sodium chloride.

Since Fourneau, in February, 1904, and Chaput, in May,

1904, read papers upon its use before medical societies, pure stovaine has been much used, and certainly since its admixture with strychnine accidents have been fewer.

Dr. A. Ash, after an experience of its use in 450 cases, speaks well of tropo-cocaine in small doses and weak solutions—a good opinion, supported by Tomaschowski. The scheme has been further elaborated by mixing the active agent with some substance of much less diffusibility than cerebro-spinal fluid, so that the anæsthetic agent might be kept localized, and the extent of its action thereby controlled and limited. For this purpose dextrine and glucose have been used, but as little of them as possible, as they tend to make the anæsthesia short, while gravity and force of injection are combated by keeping the head and pelvis raised, and the troublesome complications and deaths so frequent in the early days of spinal anæsthesia have been much lessened, and the addition of strychnine to either combination has still further made for safety.

McGavin and Williams record 250 cases in which they used stovaine-glucose, with one death from heart failure on the operating-table and one from uræmia three days after operation.

In addition to the serious consideration of what is the safest, and therefore best, analgesic to use, the position of the patient, the technique of the little operation, the dose of the analgesic, the method of modifying it for age, the part of the body to be made insensitive, and the length of time required for the operation, are all matters which have engaged the earnest and exhaustive attention of those who during the last few years have sought to obtain anæsthesia by the spinal method.

Unless such a necessity as obesity forbids, the left lateral position, with the spine well bent, is that usually chosen; if the sitting position is allowed, the anæsthesia may be confined to the lower limbs only. The routine site of puncture is in the third interspace, as already described.

This site requires the largest volume of the solution, but the anæsthesia exists the longest; the higher the puncture is made, the shorter is the resulting anæsthesia. If anæsthesia is required high in the abdomen, the second interspace must be chosen; and if in the upper limbs, or head, or neck, the second dorsal interspace is necessary, the head being lowered for a minute or two if it does not become anæsthetized. The fourth lumbar interspace is only used for anæsthesia of the perinæum, external genitals, and lower limbs. Joannesco advises the interspace between the twelfth dorsal and first lumbar vertebræ for the abdomen and lower limbs, and it is easily made out, owing to the large space which separates these two spinous processes. The stilette should be sharply withdrawn from the needle, and the solution as quickly injected, the patient turned at once on his back, and the pelvis and shoulders at once raised.

The stovaine-glucose solution is now in greater use in this country than tropo-cocaine. Its composition is: stovaine, which is not a cardiac depressant, and glucose, each $2\frac{1}{2}$ per cent., with water to 100. Sulphide or hydrochloride of strychnine is added to either stovaine or novococaine, with the idea of preventing respiratory paralysis, the dose varying from $\frac{1}{3}$ milligramme ($\frac{1}{200}$ grain) in infants to 1 milligramme in adults, and the addition is probably in some cases beneficial; but this dosage seems dangerous, as two deaths have occurred under its use. In one of these cases 1 milligramme of hydrochloride of strychnine, with 10 milligrammes of novo-cocaine, in 3 c.c. of solution, and in the other a similar quantity of strychnine and analgesic, but stovaine instead of novococaine, were used. In both the analgesic was administered in more than safe doses, and probably it was the same with the strychnine, the depressing effect upon the medullary centres of the former being too great to be overcome by the tonic power of the latter, although the dose of the

strychnine was in one of the cases sufficient to cause rigidity of the muscles of the arms and chest.

It is a question whether high anæsthesia is justifiable, respiratory embarrassment being so possible and highly dangerous, and in any person not a child the great dread of approaching death may cause unspeakable torture. If practised, it must be with strychnine, which has the disadvantage of shortening the duration of the anæsthesia, and is unnecessary in low operations.

To give food to children during spinal anæsthesia to keep them still seems unwise, as some retching, and often actual vomiting, are so liable.

The deep reflexes are the first to feel the effects of the anæsthetic; then sense of pain becomes lost; afterwards the skin reflexes fail to respond to stimuli, the scrotal being the last to do so. If motor power returns before sensation, we know that the anæsthesia is passing off, and if the operation is not finished a second injection has become necessary. In a very few cases the anæsthesia is incomplete, or it cannot be obtained at all, and an inhalant anæsthetic has to be administered. Failure to produce anæsthesia is mostly due to the injected fluid not having entered the subarachnoid space, or to insufficient dosage; in either case a further full dose may be given in ten or fifteen minutes. If in half an hour the anæsthesia has only reached the groins, two-thirds of a dose may be given, or a full dose if one hour has elapsed. With puncture in the third lumbar interspace the resulting anæsthesia is, as a rule, on a level with the point of injection; but it may reach as far upwards as the nipples, or even of the clavicles. But the hope that danger would not be associated with spinal anæsthesia has not been fulfilled, many deaths having followed its use, and many persons who eventually recovered have given their medical attendants the gravest concern; it has been followed in other cases by various forms of paralysis, and in many

diseases and conditions it is absolutely forbidden. Analgesia to the level of the fourth dorsal vertebra is the limit of safety, but when it only reaches so far pain is not abolished in abdominal operations, during which the necessary manipulations of stomach, intestines, or uterus, may cause the patient to complain of dragging sensations. The more rapid the analgesia—that is, if it is produced in from two to five minutes—the higher will it ascend, and the more likely will the most serious phenomenon of spinal anæsthesia, respiratory distress, show itself. An intense desire for more air is possible with any dosage that is calculated to produce anæsthesia, but it is more common when the dose has exceeded 6 c.c. ; on the other hand, with 8 c.c. it may not occur. When the anæsthesia reaches the level of the first, or even of the second, dorsal vertebra, it is not unusual for the patient to experience great difficulty in breathing ; the intercostal muscles are for the time paralyzed, respiration is diaphragmatic, and the danger is extreme for the next fifteen or twenty minutes. Fortunately, a fatal issue is rare.

The feeling of air-hunger seems to depend upon the analgesic affecting the respiratory centres in the medulla, through its introduction into the system, and not upon the fact of its injection into the spinal subarachnoid space. The author observed the condition in an extreme degree in a case which was only too nearly fatal, in which cocaine had been injected into the gums preparatory to teeth-extraction. Other symptoms noticed at the same time were extreme dilatation of the pupils, great apprehension of approaching death, cold sweats, intense pallor, and small, thready pulse ; indeed, the radial pulse was for a time not perceptible. The remedies applied were artificial respiration, inhalation of oxygen, hypodermic injections of strychnine, and the internal administration of brandy; and of these, the oxygen gave most relief : the patient afterwards said that to it only she owed her life.

Some children whimper from time to time during spinal anæsthesia; others are restless or move uneasily when portions of the bowel during abdominal operations are drawn upwards; others exhibit no sign of pain or discomfort. This absence of pain impressed the writer very strongly on seeing a three-year-old child bear unflinchingly strongly-applied over-correction that was necessary after an operation for club-foot. But whether patients thus anæsthetized lie motionless or are restless, they all, so far as the writer has observed, give evidence of being under a deleterious influence in their pallor and widely dilated pupils.

Frequently in from five to fifteen minutes after the injection of from 2 to 6 c.c. of cocaine solution, pallor, sweating, nausea, vomiting, and occasionally marked apprehension, set in. Stovaine in similar dosage is less likely than cocaine to cause these troubles; but it has its particular drawbacks—happily capable of being greatly reduced by its dilution with glucose or dextrine—in causing relaxation of the sphincter ani, retention of urine, dysuria, engorgement of the penial vessels, and various forms of paralysis. Tropo-cocaine is not followed by the latter consequences, and is less likely than either cocaine or stovaine to produce the former. Whatever drug be chosen, it should be fresh.

Hardonier reports a fatal issue after nine successful cases, the patient dying fifteen minutes after an operation for the relief of strangulated hernia had been begun, and a month later he had to record other fatal cases.

Headache, with a slight rise of temperature and some sickness or retching—the younger the patient, the less is the tendency to vomit—during operation or the day afterwards, are the common and unimportant incidents of spinal anæsthesia, and in the same class is the slight pallor of face, while the lips continue red. The more important are pallor, with cyanosis of the lips, dyspnœa, and weakened

respirations—symptoms indicating that those injurious effects upon heart and lungs are produced which it was hoped would be avoided.

Untoward symptoms may be termed *immediate* when they arise during or shortly after the anæsthesia; they include marked pallor, cold sweats, apprehension of death, small and quick pulse, dilated pupils, and air-hunger. All of them may be assumed to be due to the analgesic *per se;* while the *secondary*, or *post-analgesic*, phenomena, as headache, continued vomiting or retching, and febrile temperature, retention of urine or dysuria on several days succeeding the operation, and various paralyses, may be ascribed to the analgesic having been injected into the spinal theca, and particularly so when the paralysis assumes a permanent form. Other conditions having a similar origin are severe pain localized down the spine (rachialgia), paræsthesic sensations, as formication and numbness, and suppurative spinal meningitis.

While spinal anæsthesia does not seem likely at present to displace to any great extent inhalant anæsthetics, it has advantages over the latter in not causing post-anæsthesic pneumonia and acute bronchitis, or, when the anæsthesia is absolute, operative shock; in usually enabling food to be given shortly after the operation is finished; and it is the preferable anæsthetic in cardiac, respiratory, and renal disease; is well borne by persons addicted to alcohol, and eagerly accepted as the alternative by those who fear absolute unconsciousness. All are agreed that it is of very particular value in operations for acute intestinal obstruction; it is said not to be followed by paralytic states of the intestines in case of peritonitis; is very useful and often the only possible form of anæsthesia in instrumental delivery in women suffering from acute respiratory disease, special precautions being taken to cope with the almost certain post-partum hæmorrhage that follows.

The drawbacks to its use are that the head, neck,

and upper part of the trunk can only be anæsthetized by passing the limit of safety; that it does not act in some persons at all, and is attended, as we have observed, by dangerous symptoms in others, either during the period of anæsthesia or afterwards, and because experience has taught us these dangerous or untoward symptoms are very likely to occur in quite a large number of ailments, both functional and organic.

It is contra-indicated in long operations, because, apart from pain, a person cannot be kept absolutely quiet for any length of time, and in difficult ones because it may be unwise to have the person conscious of what is being done; when any present or past cerebral disease suggests the possibility of the foramen of Magendie being closed by adhesions or the pressure of tumours; in those who dislike spinal anæsthesia or are frightened at the sight of blood or cutting; in diabetics who require amputation of the lower limbs for gangrene; in operations on the pelvis, when the latter requires to be kept raised; in acute infectious diseases, as suppurative meningitis has been known to follow; in operations on tendons in spastic paralysis, as the relaxation of muscles is so great as to be misleading; in scoliosis in children under fifteen years of age; in non-compensated cardiac diseases and pericarditis with effusion; in acute and chronic diseases of the spinal cord; in spinal curvature; in vascular sclerosis and aneurisms of cerebral vessels; and in all conditions in which the withdrawal of more than a very small quantity of cerebro-spinal fluid has been necessary, and certainly in all who cannot remain in bed a good twenty-four hours after anæsthesia. Some surgeons, particularly Continental ones, also include atheromatous conditions of the arteries, recent syphilis, and a high temperature; but a few do not.

If the attending doctor is new to his patient and is told that he has had post-basic or sporadic cerebro-spinal meningitis, it will be well if that doctor respects and

accepts the diagnosis of his predecessor, and administers an inhalant; or he may find, to his annoyance, that no cerebro-spinal fluid will flow, through adhesions having closed the subarachnoid space. One may also suggest that empyema, and particularly double empyema, at all ages should certainly be included amongst the contra-indications, as, one or both lungs being already embarrassed, the advent of intercostal paralysis would necessarily place the patient in the gravest danger; and, for similar reasons, abscess under the diaphragm or in the upper abdomen should be excluded; yet in many of these cases it is constantly used, both in this country and abroad.

When paralysis occurs, it is observed in from nine to thirteen days after injection, and for the most part is limited to the urinary organs or ocular muscles, and of the latter usually the external rectus. Paraplegia has been recorded.

Spielmayer has examined histologically the nervous systems of several people who died some time after the injection of stovaine. In one old woman whose death seemed directly due to the injection he found chromolytic changes of the ganglion cells distributed all over the nervous system. In three other cases the polygonal cells of the spinal cord were swollen and rounded. In other cases of death no abnormality has been noticed in the nerve roots, or the spinal cord, or its membranes.

Treatment of Complications.—Sweating, pallor, and feeble pulse may be combated by small doses of brandy; but when these conditions are associated with extreme respiratory embarrassment, hypodermic injections of strychnine, artificial respiration, and inhalations of oxygen, may all be required, and perhaps prove futile.

Therapeusis.—The value placed upon lumbar puncture in the treatment of tubercular meningitis, the purpose for which it was originally introduced, has not been upheld

by those who have since practised it, but it is still sometimes used, and is said to be most useful when employed during the first week of the illness; and although few cures can be ascribed to its use, it is said to relieve such urgent symptoms as headache, general discomfort, convulsions, etc.; but the relief obtained does not last many hours, and the operation may require to be frequently repeated.

The idea of establishing permanent drainage at the lower part of the spinal canal in suitable cases has been carried into practice, but up to the present time has not been followed by satisfactory results.

While lumbar puncture and the withdrawal of 15 to 20 c.c. of cerebro-spinal fluid—much more might subject the bulb to pressure and bring about a rapidly fatal issue—has fallen into comparative desuetude as a curative process, that procedure plus the injection afterwards of medicinal agents has been proved to be of the greatest value, as in the case of meningococcal serum, collargol, lysol, etc., in epidemic cerebro-spinal meningitis, the bactericidal remedy being thus brought into immediate contact with the bacilli it is intended to destroy. The experience gained in the epidemics in Silesia, America, Lisbon, Glasgow, Edinburgh, and Belfast, with one or two notable exceptions, confirms the opinion expressed at the meeting of the British Medical Association at Sheffield in 1908—that intrathecal medication is by far the most important treatment as yet known for this very fatal disease.

Spinal must not be confused with sacral anæsthesia, in which puncture is made between the coccyx and pelvis, and the injected fluid does not pass within the dura, but is distributed between the sacral periosteum and the dura mater, the nose being painted at the same time with a strong solution of cocaine. This form of anæsthesia has been practised during labour, but the duration of the

anæsthesia is limited, and in a number of cases there is no result.

Chorea and tetanus have been successfully treated by daily intrathecal injections of 2·5 c.c. (about 40 minims) of a 25 per cent. solution of sulphate of magnesia, boiled in a previously sterilized test-tube. In tetanus repeat as often as four-hourly if the return of tetanic spasm compels, and as long as may be required, the usual result being that the spasm becomes altogether relaxed in a very short time. Extensive paralysis, profuse sweating, and the accumulation of much mucus in the bronchi, but not purgation, may succeed each injection and last some time. The head should be raised, for the reasons given under Spinal Anæsthesia.

The great benefit of this form of treatment has been demonstrated in cases of tetanus in which full and frequent doses of bromide of potassium and chloral hydrate, with subcutaneous injections of tetanic antitoxin, had produced no result. If it is found that the above dosage only relieves the patient for a few hours, 1 drachm of the solution may be used in a robust adult with more lasting, and perhaps permanent, benefit.

In cases of fracture of the skull the removal of cerebro-spinal fluid from the thecal cavity has caused the final disappearance of coma, and given great relief to severe pain.

APPENDIX B

THE NASAL ACCESSORY SINUSES

We have many times pointed to diseases of the nose and its accessory cavities as the possible but rare causes of intracranial abscess, purulent meningitis, and cavernous sinus thrombosis; it is, therefore, proceeding on the principle that prevention is better than cure, within my rôle as a writer upon these troubles to indicate the symptoms which these causal diseases exhibit, so that, their presence having been ascertained, their treatment and cure may become possible and the graver diseases be prevented. The less often a disease occurs, the more likely is it to pass without correct diagnosis.

The orbit is completely surrounded, except in its outer wall, by the large air spaces connected with the upper respiratory passages, which are non-existent in children, except in the case of the maxillary, and are formed after puberty by the absorption of cancellous bone; they communicate with the nasal cavities by minute openings, which are lined with mucous membrane continuous with that of these cavities.

These spaces, or cavities, are divisible into an anterior and posterior group. The former comprises the antrum of Highmore, the frontal sinus, and anterior ethmoidal cells, all of which open into the middle meatus of the nose; and as by this route the larger stream of air passes, disease of the

anterior group is the more common. Inflammation and suppuration in them affect the eye directly by compression and displacement. The posterior group includes the sphenoidal and the posterior ethmoidal cells, which empty into the superior meatus, by which the smaller stream of air passes, and this group is therefore less commonly found inflamed or suppurating. The following associations are common : fronto-maxillary and fronto-anterior ethmoidal sinusitis and sphenoidal posterior ethmoidal sinusitis.

As bearing upon cavernous sinus thrombosis, it must be remembered that all the air spaces about the orbit have a venous return, which enters the orbit and is continued by the ophthalmic veins, together or singly, into the cavernous sinus.

When a fœtid purulent discharge from the nose cannot be accounted for by changes in the colour and thickness of its mucous membrane, or changes in the colour and bulk of the anterior ends of the inferior or middle turbinated bones, and there is no polypus in the nose, or adenoids in the naso-pharynx, there is *primâ facie* evidence that the discharge is due to disease of the nasal accessory sinuses. Such disease is more frequent in men than women, and is absent in children for the reason already advanced. The infective micro-organism is the *Micrococcus catarrhalis*, introduced probably by inhalation.

Diseases of the frontal and maxillary sinuses may be comparatively easy of diagnosis ; those of the ethmoidal and sphenoidal sinuses will probably always present difficulties even to those whom constant practice in posterior rhinoscopy has made more efficient than others.

The functions of the frontal and maxillary sinuses are not connected with the sense of smell, but the bones in in which they ocur being as light as possible, they provide more room for the resonance of the air in vocalizing.

The frequency of suppuration in the accessory cavities

is in the following order : maxillary, frontal, ethmoidal, sphenoidal.

The order of frequency as causes of acute cerebral disease is : frontal, ethmoidal, sphenoidal, maxillary.

If there is no nasal discharge it does not imply the absence of suppuration in the air cells, the orifice of which may be closed up and an empyema be present ; the mouths of the sinuses, except the frontal, are badly situated for drainage by gravitation.

Frontal Sinus.—There is often a temporary closure of the funnel-shaped cavity, the infundibulum, which leads from the nose into the frontal sinus, by extension of the swelling of the nasal mucous membrane to that of the infundibulum.

Any of us may from time to time have an opportunity of observing the shutting off of the frontal sinus from the nose in such a case as the following : Early in January, 1909, I saw a young man, twenty-one years of age, who was suffering from influenza, of which the more prominent symptoms were bronchitis, general headache, pains in the limbs and at the back of the eyes, slight pyrexia, and a very troublesome coryza. After being ill a week he began to complain of pain and tenderness in the forehead, with fulness and weight just above the bridge of the nose. These troubles increased in severity until they became almost intolerable, and sleep, even after fairly large doses of morphia and sulphonal, was impossible. I surmised the cause, and suggested constant hot fomentations and frequent hot inhalations. After the condition had lasted three or four days, on the patient blowing his nose with some force, there occurred a sudden discharge of about half an ounce of a watery flux. Immediate relief followed. Neuralgia on the course of the supra-orbital nerve is usual, and often returns at the same time each day, but it was not exhibited in the above case.

Such a desirable termination does not always happen,

and distension of a frontal sinus may be so great as to cause proptosis and displacement of the corresponding eyeball, the trouble being usually confined to one side ; it may become necessary to reopen the closed neck of the infundibulum with probe or trocar, which is said to be a particularly difficult piece of work, and which the author has never seen occasion to attempt.

When the secretion of the inflamed mucous membrane can no longer pass into the nose, suppuration eventually results, and if not relieved the ultimate consequences will be destruction of the mucous membrane and denudation of the periosteum, ending in necrosis of the bone. If the channel again becomes pervious, a foul nasal discharge is established, revealable by rhinoscopic examination as entering the nose by the middle meatus ; but such knowledge of affairs does not mean an exact diagnosis, as pus in that *locale* is common also to suppuration in the ethmoidal cells and the maxillary sinus. The most frequent course of events is that the channel into the nose remains more or less occluded, and the walls of the cavity, where necrosed, give way ; if it is the supra-orbital plate of the frontal bone, which it usually is, pus forces its way through the inner half of the upper eyelid. If it is the posterior wall of the frontal sinus that becomes necrosed and perforated, three associated or separate results may ensue—**pachymeningitis,** from irritation by necrosed bone; **leptomeningitis,** if the micro-organisms contained in the pus pass through the dura and reach the pia-arachnoid; and **cerebral abscess,** if the same are carried into the brain.

The Maxillary Sinus.—The maxillary sinus, or antrum of Highmore, is a possible source of cerebral infection, but the writer cannot call to mind having heard or read of such a cause having actually been traced. Reference is only made to it in order that disease in it may be the more readily differentiated from that in other sinuses.

The orifice of the antrum is situated in the hinder part of

the infundibulum, and is common with that of the anterior ethmoidal cells, or a little posterior to it, beneath the middle turbinated bone; and as this orifice is nearer the roof than the floor of the antrum, escape of its secretion is not favourable, and decomposition of the retained contents may readily take place if this orifice becomes temporarily blocked with mucus.

The symptoms are swelling and œdema of the cheek, an *intermittent* fœtid nasal discharge often occurring when the patient holds his head down, the discharge often finding its way, particularly at night-time, into the posterior nares; and the patient complains of a disagreeable taste in his throat, and is often compelled to hawk matter from the naso-pharynx; the pain is one-sided, but not so periodic as in the case of the frontal sinus.

On rhinoscopic examination, after the interior of the nose has been suitably cleansed and the patient has been directed to bend his head forward, pus may perhaps be seen to trickle from the middle meatus; but as such trickling is common to suppuration in other cavities, other differentiation becomes necessary, and this is effected in case of the frontal sinus by making the patient lie prone on a table, with his head hanging over the edge. If just afterwards pus is found in the middle meatus, the frontal sinus is eliminated as the source of the pus.

Additional assistance in diagnosis may be obtained by transillumination. The patient being in a darkened room, a small electric lamp of about five candle-power is introduced into his mouth. If there is much pus a dark shadow will be present below the eye on the diseased side, and a bright crescent of light in the same position and a red glare of the pupil if there is no pus in the antrum.

The Ethmoidal Sinus.—The ethmoid bone (ἠθμός, a sieve) is a light spongy bone of cubical shape, situated at the anterior part of the base of the skull, between the orbits at the root of the nose. The salient points of the

ethmoidal bone which are pertinent to us in the matter under consideration are that the lateral masses or ethmoidal cells, which constitute the bone, consist of a number of thin-walled cellular cavities, divided by transverse bony partitions into **anterior, middle,** and **posterior** cells, which do not communicate with each other. These cellular cavities are placed between two vertical plates of bone, the outer one of which, the **os planum**, forms part of the inner wall of the orbit, and the inner one, which terminates below in a free convoluted margin, forms part of the outer wall of the nasal fossa of the same side. The middle turbinated or spongy bone, which, when necrosed, requires removal in the treatment of ethmoidal abscess, in the back part of which bone there is a narrow oblique fissure, the superior meatus of the nose, into which the posterior cells open, is bounded above by the superior turbinated bone.

The anterior cells communicate with the frontal sinus above and the middle meatus below, per the infundibulum; the middle cells also open into the same meatus, and pus found there by posterior rhinoscopy indicates either ethmoidal abscess or frontal sinus abscess.

The origin of ethmoiditis, the forerunner of abscess, is frequently catarrhal, as in the case of coryza from an influenzal or common cold, measles and scarlatina, traumatism of any kind, and syphilis; the disease is generally on both sides, and occurs chiefly in early manhood. The inflammation begins in the muco-periosteum lining those processes of the ethmoid which dip into the nasal fossæ.

The symptoms while the disease is yet in the inflammatory stage are pain between the eyes, loss of smell, excess of nasal secretion, swelling of the glabella, and swelling and discoloration of the eyelids. When abscess results, the most dependable signs are objective, as already stated; the early symptoms continue, and we find secretion has taken on a fœtid character; the bridge of the nose

broadens from the abscess opening into the nose, or it may open into the upper eyelid or below the internal canthus.

Suppuration in the ethmoidal cells may cause neuralgia, apparently intractable, but it ceases altogether when the pus is given free escape and the necrosed bone removed.

According to Dr. Woakes, the usual result of abscess in this sinus is necrosis, which may advance to the orbital plate of the frontal bone, causing purulent meningitis, etc., or progress to the superior maxilla, producing abscess of the maxillary sinus, and he emphasizes the fact that this necrosing ethmoiditis is very likely to be the forerunner of nasal polypi.

The Sphenoidal Sinuses.—These sinuses are situated in the body of the sphenoid bone, on the posterior part of the roof of the nasal fossæ, and are separated by an irregular bony septum, and above them lies the optic groove, for the accommodation of the optic commissure. They open on each side into the superior meatus with the posterior ethmoidal cells, with the inflammation or suppuration of which they are usually associated. They are of some size at six years of age, but not fully developed until after puberty. The causes of disease in them are chiefly catarrhal. The symptoms observed are a deterioration of the general health, possibly vomiting, pain of a general character, or periodic and localized in the occiput, less often in the forehead, but it may be absent. Possibly there is loss of sense of smell, or the patient experiences a very bad odour; there is feverishness, except in chronic cases; there are also noises in the head, dizziness, and a fœtid discharge, emptying viâ the superior meatus into the naso-pharynx, and therefore not always seen on examination, but causing a persistent coughing and efforts at expectoration; and, owing to the nearness of the optic nerve, optic neuritis occurs unless the disease runs an extremely rapid course to a fatal issue.

In women a chlorotic hue may be present in case of suppuration in any of the accessory cavities, but in the sphenoidal sinuses this hue may become an intensely livid pallor.

Disease of this sinus may produce post-basic meningitis, or thrombosis, or suppuration in the cavernous sinus.[1] If thrombosis occurs, the objective signs of that condition will be in evidence; if suppuration, a general pyæmic condition would ensue, and the true state of affairs only be recognized in case of a post-mortem examination.

Prognosis.—The outlook is favourable, unless infective thrombosis of the cavernous sinus develops, after which hope must cease.

Diagnosis.—Pain is in the forehead or in the little hollow between the eyebrows in frontal and ethmoidal, in the cheek in antral, and mostly in the occiput in sphenoidal sinusitis. If on posterior rhinoscopy pus is seen to trickle into the middle meatus, it comes either from the antrum or frontal and anterior ethmoidal cells; if pus is seen to pass into the superior meatus, it comes from the posterior group of sinuses.

Treatment.—Fomentations and hot inhalations containing small quantities of ol. eucalypti and menthol. If suppuration occurs in the frontal sinus, and the infundibulum is not patent, an opening may be made in the forehead. Ethmoidal and sphenoidal suppuration require the knowledge and skill of the expert for their treatment.

The treatment of antral suppuration is doubtless so well known that it does not require repeating here.

[1] *Vide British Medical Journal*, p. 771, September 29, 1906.

REFERENCES ADDITIONAL TO THOSE IN THE FOOTNOTES OR TEXT

GOWERS, SIR WILLIAM : 'Diseases of the Nervous System', vol. ii. Churchill, 1893.

BASTIAN : 'Paralyses—General, Bulbar, and Spinal.' Lewis, London, 1886.

HERMANN : Osler and Macrae's 'System of Medicine,' vol. vii. 1910.

BALL : 'Diseases of the Nose.' Baillière, Tindall and Cox, 1894.

Guy's Hospital Reports, 'Cerebral Pneumonia,' p. 65, Series I vol. ii.

HAYWARD, WARRINGTON : 'Phlebitis and Thrombosis.' 1906.

TAYLOR, JAMES : 'The Nervous Diseases of Children.'

HAMILTON : 'Pathology, Systematic and Practical.' 1894.

NEUMANN : 'Otitic Cerebellar Abscess,' edited by Lake. Lewis, London, 1909.

GORDON, KNYVETT : *Practitioner*, November, 1909.

SCOTT, SIDNEY : *Practitioner*, January, 1910.

BROADBENT : 'Syphilitic Pachymeningitis of Cerebellar Fossæ,' *Lancet*, vol. i., p. 186, 1882.

BROADBENT : 'Pachymeningitis,' *Lancet*, vol. i., p. 106, 1884.

BROADBENT : 'Transitory Pneumonic Aphasia,' *Lancet*, February 17, 1894.

RUSSELL, RISIEN : 'The Cerebellum and its Affections,' *British Medical Journal*, February 26, 1910.

GARBETT : 'Death from Injection of Novo-cocaine and Strychnine,' *British Medical Journal*, March 19, 1910.

MANKIN, GUTHRIE : 'Meningitis in Children,' *British Medical Journal*, April 30, 1910.

LANDON, STIRLING : 'Lumbar Puncture in Meningitis and Allied Conditions,' *Lancet*, April 16, 1910.

LANDON, STIRLING : 'Méningisme,' *British Medical Journal*, Epitome No. 280, May 21, 1910.

LANDON, STIRLING : 'Influenza, with Meningitis as a Complication,' *Lancet*, p. 615, March 2, 1901.

FORTH : 'Various Forms of Meningitis,' *British Medical Journal*, October 21, 1895.

MACEWEN, SIR WILLIAM : 'Pyogenic Diseases of the Brain and Spinal Cord.'

MACKENZIE : 'On the Diseases of the Eye.' Longmans, Green and Co., London, 1840.

SAUNDBY : 'Cerebral Influenza,' *British Medical Journal*, October 21, 1895.

CURL : 'Blood Changes in Meningitis in Children,' *Lancet*, May 6, 1905.

CURL : 'Bacteriology of Simple Post-basic Meningitis,' *British Medical Journal*, October 23, 1897.

THURSFIELD : 'Post-basic Meningitis,' *Lancet*, February 16, 1901.

THURSFIELD : 'Recovery from Tuberculous Meningitis,' *Lancet*, June 10, 1905.

MOTT : 'Cerebro-spinal Fluid in Relation to Nervous Diseases,' *British Medical Journal*, December 10, 1904.

ORMEROD : 'Cerebro-spinal Meningitis,' *Lancet*, April 29, 1905.

ORMEROD : 'Purulent Cerebro-spinal Meningitis due to Typhoid Bacillus,' *British Medical Journal*, April 25, Epitome No. 217, 1908.

SYMONS, ST. CLAIR : 'Spinal Anæsthesia,' *Lancet*, August 15, 1908.

SUDABINI : 'Spinal Anæsthesia,' *Lancet*, October 24, 1908.

GRAY : 'Spinal Anæsthesia in Children and Infants,' *Lancet*, September 25, 1909.

JOANNESCO, PROFESSOR : 'General Spinal Anæsthesia,' *British Medical Journal*, November 13, 1909.

WHITE, MAINWARING : 'Sinusitis,' *Lancet*, December 6, 1908.

WHITE, MAINWARING : 'Sulphate of Magnesia in Tetanus,' *British Medical Journal*, Epitome No. 297, June 5, 1909.

MOTT : 'On Diagnosis of Syphilitic Diseases in the Nervous System,' *British Medical Journal*, June 2, 1909.

LARKIN : 'Cerebro-spinal Fever,' *Practitioner*, June, 1909.

SHORSTEIN : 'Abscess of Brain in Association with Pulmonary Disease,' *Lancet*, October 19, 1909.

BARR, DR. : 'Diseases of the Ear.' Maclehose and Sons, 1884.

FUCHS, DR. ERNEST : 'Textbook of Ophthalmology' ; translated by Dr. Duane. D. Appleton and Co., New York, 1901.

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